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DEPARTMENT OF REVIEWS

The Journal will make an especial feature of the review of monographs and books bearing upon the field of Internal Medicine. Authors and publishers wishing to subject such material for the purposes of review should send it to the editor. While obviously impossible to make extended reviews of all material, an acknowledgment of all matter sent will be made in the department of reviews.

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Insulin and Carbohydrate Tolerance*

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INTRODUCTION

SINCE the discovery of insulin by Banting (1), there has been a great deal written concerning the cure of diabetes mellitus with this new therapeutic agent. MacCallum (2), Boyd and Robinson (3) have given us some meagre hope of a cure with microscopical evidence in favor of the regeneration of the islands of Langerhans; while Newburgh (4), Harrison (5), and others (6, 7) present clinical evidence of a negative nature concerning a cure. With this controversy still unsettled, five cases were carefully selected from our diabetic clinic with this question ever present. Is there any evidence of a cure, or even a partial cure of human diabetes mellitus as shown by the tolerance for glucose after insulin has been used daily over a long period of time?

METHOD OF PROCEDURE

The high fat, low protein, low carbohydrate weighed diets of Newburgh (8) and Marsh were used throughout, and the insulin was adjusted to keep the urine just free from any reducing substance and ketone bodies. The observations reported later in this paper were made on five patients who had been receiving insulin daily, and had been observed at different intervals at the clinic over long periods of time, varying from 32 to 45 months. Observations on these five patients provide data as to whether or not insulin improves the tolerance for glucose of each patient, when the treatment is prolonged over many months. By tolerance for glucose is meant the total glucose content of a diet that a patient can take without glycosuria. The total glucose content of any diet is derived by the formula: 100% of the carbohydrate plus 58% of the protein plus 10% of the fat (9). The blood sugar estimations were made by the Folin and Wu method. Benedict's quantitative and qualitative methods have been used for sugar. Gerhardt's ferric chloride test for diacetic acid, and the nitroprusside test for acetone in the urine. The patients were weighed, when possible, to the nearest kilogram without clothes. If weighed with clothes, due allowance was noted and made.

For insulin injection one cubic centimeter syringes, which were calibrated in twentieths or hundredths of a cubic centimeter, were used. Dosage is recorded throughout as so many units. The drug was administered subdermally two or three times a day. Insulin

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was given routinely, unless otherwise specified, at 7 A. M.; 11:30 A. M., and 5 P. M. each day.

Five patients have been kept under observation for periods of 32 to 45 months, each on a diabetic diet suitable for that particular individual and with enough insulin each day to keep the urine aglycosuric and aketonuric. Upon their initial visit each patient was studied at length in the hospital, at which time their glucose tolerance was determined. Each has been restudied in the hospital as well as during other subsequent visits to the Diabetic Out-Patient clinic, at which time glucose tolerance redeterminations were made.

These five patients form a small group, who have distinguished themselves for their loyalty, honesty, intelligence and long period of observation. They were accordingly selected for this study because we were justified in believing that we would get reliable information from them. At the beginning of the investigation, which was undertaken some two years ago, it was not known whether the subsequent treatment with insulin would give evidence of improvement or the converse. As already mentioned, these five cases are used because of their reliability and because they have continued to adhere strictly to our plan to the end of our study. The glucose tolerance of each individual is always determined by beginning with bed rest and a daily diet consisting of protein 20 grams, fat 86 grams, and carbohydrate 15 grams, without insulin. The total glucose content of this diet is 30 grams. After an aglycosuric period of three to four days, each foodstuff is proportionately increased so that the next diet has a total glucose content of 50 grams and consists of protein 28 grams, fat 129 grams, and carbohydrate 20 grams. Similarly, after aglycosuric periods of the same time length, the diet is raised proportionately until its total glucose content is 70 grams, consisting of protein 34 grams, fat 163 grams, carbohydrate 29 grams. The fourth level affords 90 grams of total glucose, consisting of protein 54 grams, fat 210 grams, and carbohydrate 37 grams. To the latter diet carbohydrate is added in ten gram measures every third or fourth day, until there is a glycosuria.

It will be found that at a certain place in this plan each diabetic will have glycosuria. Any part of this regime that he is able to use just without glycosuria measures his complete ability to burn glucose, (a pancreatic function), or his glucose tolerance. This point in the dietary scheme in moderately severe, or severe diabetics is usually so low in calories that the patient would have to live on a submaintenance diet. Of course, this is a definite indication for a proportionate increase in the amount of each of the foodstuffs to make up a maintenance diet for that particular individual. Since glycosuria would occur with additional food, insulin in proportionate doses to keep the urine aglycosuric and yet prevent hypoglycemic reactions is given. This has been the procedure in each of the five cases.

Report of Cases

Case I. L. C., schoolgirl, age 18, first complained of polyuria, polydipsia, polyphagia, in June, 1922. She entered the hos-

pital in September of the same year, weighing 37 kilograms. The urine gave strongly positive tests for glucose and diacetic acid, and a fasting blood sugar was 0.38%. It was found that she could tolerate without insulin and just without glycosuria a diet of protein 54 grams, fat 214 grams, and carbohydrate 38 grams, which has a total glucose content of 90 grams. This amount was her original glucose tolerance.

In January, 1923, she returned to the clinic because of infection and uncontrollable glycosuria. After both had entirely disappeared, it was found that now she could only tolerate without insulin, a diet composed of protein 31 grams, fat 176 grams, and carbohydrate 28 grams, which contained 61 grams of glucose, the new level of glucose tolerance. Weight at this time was 39.1 kilograms.

In May, 1923, because of uncontrollable glycosuria of unknown cause, she returned to the clinic, weighing 34.9 kilograms and with the next succeeding twenty-four hour urine specimen containing 5.3 grams of glucose. After aglycosuria was established it was noted that now a diet of protein 28 grams, fat 129 grams, and carbohydrate 20 grams, with a total glucose content of 46 grams could be tolerated without insulin and glycosuria. It was found that this diet contained insufficient calories (1368) for an active girl of her age. Insulin and a maintenance diet of protein 55 grams, fat 220 grams, and carbohydrate 35 grams were prescribed in June, 1923. At first a daily total of 9(5-4) units of insulin in divided doses were required to keep her urine aglycosuric.

Because of infection, coryza, and tonsillitis, she returned to the clinic October, 1923. She was discharged in December of the same year, with a diet of protein 55 grams, fat 220 grams and carbohydrate 40 grams, and with a total of 59 (22-14-23) units of insulin in divided doses during the day. Weight then was 42.5 kilo.

This patient next revisited the clinic January 1st, 1926, because of glycosuria, hypoglycemic reactions and head cold. The next twenty-four hour urine specimen contained 34.4 grams of glucose and a fasting blood sugar was .222%. After aglycosuria had been established, it was found that she could tolerate a diet of protein 55 grams, fat 220 grams and carbohydrate 35 grams, with a daily dose of 56 units of insulin. It was found that by spacing the insulin dosage differently, that is, 32 units at 7 A.M., 20 at 5 P.M., and 4 at 10 P.M., the urine would remain aglycosuric at all times. Discharge weight was 54.5 kilos. She has been aglycosuric and without insulin reactions on this regime for the past four months.

Case II. R. B., contractor, age 28, first noted increased thirst and urination with loss of weight in February, 1922. He entered the clinic October, 1922, with a weight of 53.9 kilos and a urine, which gave strongly positive tests for glucose and diacetic acid. It was found that he could tolerate, without insulin and just without glycosuria, a diet composed of protein 40 grams, fat 180 grams, and carbohydrate 30 grams. This diet contained a total glucose of 71 grams or the original glucose tolerance.

In February, 1923, the patient returned because of uncontrollable, irregular glycosuria and infection, head cold. After the urine was rendered aglycosuric it was found that he could not tolerate, without glycosuria, a diet with a total glucose content of 64 grams composed of protein 33 grams, fat 171 grams, carbohydrate 28 grams. Thus the glucose tolerance at this time lies somewhere between 64 and 50 grams of glucose, the latter being in the next lower routine diabetic diet of protein 28 grams, fat 129 grams, and carbohydrate 20 grams. Because of the glycosuria and of the patient's occupation, age, sex, and height, (6 feet, 2 inches) it was readily observed that a maintenance diet with insulin would be necessary.

Accordingly, in March, 1923, a diet of protein 58 grams, fat 281 grams, carbohydrate 57 grams, with a daily total of 28 (14-0-14) units of insulin in divided doses during the day were prescribed to keep the urine aglycosuric. Weight was then 53 kilos. Because of a head cold the insulin dosage was raised after one month to a total of 36 (18-0-18) units in divided doses during the day. The diet was kept constant. From

May, 1923, to January, 1925, the patient was aglycosuric and free from hypoglycemic reactions on the above diet and 36 units of insulin daily.

In January, 1925, he contracted influenza and upon recovery it was found that now 48 (8-20-20) units of insulin in divided doses during the day, together with the same diet, were needed in order to maintain aglycosuria. From the latter part of January, 1925, to January, 1926, this regime was continued without glycosuria or hypoglycemic reactions. Average weight during this period was 65.9 kilograms.

In the latter part of January, 1926, this patient entered the hospital semi-comatose due to a severe upper respiratory infection. After aglycosuria had been established and the infection had been entirely cleared, he was discharged in March, 1926, on a diet of protein 60 grams, fat 280 grams and carbohydrate 60 grams, with a total of 85 (25-30-30) units of insulin in divided doses. Weight was upon discharge 60 kilograms.

Case III. R. K., telephone operator, age 18, first noticed dysuria, pruritus vulvae, loss of weight and strength, polyuria and polydipsia in January, 1924. She entered the clinic February, 1924, weighing 46.1 kilograms, with strongly positive urinary glucose and diacetic acid tests. After aglycosuria had been established, it was found that this patient could tolerate without glycosuria a diet of protein 55 grams, fat 225 grams, and carbohydrate 35 grams. This diet has a total glucose content of approximately 90 grams, which is the original glucose tolerance.

As it was decided in April, 1924, to increase the diet slightly and add insulin, this patient was given a single dose of 8 units at noon with a diet of protein 55 grams, fat 230 grams, and carbohydrate 45 grams.

During the interval between hospital visits the patient found that it was necessary, because of an irregular glycosuria, to raise the insulin to a single dose of 20 units at noon. The diet remained constant.

As this regime did not keep her aglycosuric, she returned to the clinic October, 1924, with a weight of 53 kilograms, and the urine yielding 27.1 grams of glucose in the next 24 hours. After the urine was aglycosuric, and the foodstuffs raised gradually and proportionately, it was found that she could now only tolerate without insulin and glycosuria, a diet composed of protein 34 grams, fat 164 grams, and carbohydrate 30 grams. The glucose content of this diet is approximately 70 grams, the new glucose tolerance level. The patient was discharged on the same diet of protein 55 grams, fat 230 grams, and carbohydrate 45 grams, but with a daily total of 30 (10-10-10) units of insulin in divided doses during the day.

This kept her aglycosuric and free from hyperinsulinemia reactions and infections until March, 1926. She then returned with a nasopharyngitis and uncontrollable glycosuria. At this visit a fasting blood sugar was .222% and the urine gave strongly positive tests for glucose and diacetic acid. The weight was 61 kilograms.

After the infection had been cleared and the urine rendered aglycosuric, she was discharged on exactly the same regime. On this plan she has remained both aglycosuric and free from hypoglycemic reactions to date.

Case IV. D. A. H., dietitian, age 25, first noted the symptoms of loss of weight, weakness, polyuria and polyphagia in August, 1923. At the time of entrance to the clinic, August, 1923, she weighed 45 kilograms and the urine showed strongly positive tests for glucose and diacetic acid. It was found that she could tolerate, without insulin and glycosuria, a diet of protein 54 grams, fat 210 grams, carbohydrate 37 grams, plus 30 extra grams of carbohydrate. This diet has a total glucose content of 120 grams and a caloric value of 2374 calories. Because of the patient's under-nutrition, weakness, and the insufficient caloric value of the tolerated diet, she was given in November, 1923, a diet of protein 55 grams, fat 250 grams and carbohydrate 70 grams, with a daily total of 52 (32-20) units of insulin in divided doses. In the interval between clinic visits on this regime there was a history of an occasional slight glycosuria and a hypogly-

cemic reaction, but no infections of any nature.

In April, 1925, the patient returned to the clinic weighing 59 kilograms. At this time it was found that the aforementioned diet of protein 55 grams, fat 250 grams and carbohydrate 70 grams, combined with a daily total of 52 (32-20) units of insulin in divided doses kept the urine aglycosuric.

Because of a head cold the patient revisited the clinic in August, 1925, weighing 60 kilograms. At this visit it was found that in order to avoid glycosuria and insulin reactions, the diet must consist of protein 60 grams, fat 220 grams, and carbohydrate 60 grams, with the same daily total of 52 (32-20) units of insulin in divided doses. She returned home in April, 1926, weighing on this regime 63 kilograms. She has since remained free from glycosuria and insulin reactions.

Case V. F. J., age 26, junior medical student, first complained of weakness, polyphagia, polydipsia, polyuria, and loss of weight in January, 1919. He entered the clinic in June, 1919, weighing 45 kilograms and the urine giving strongly positive tests for glucose and diacetic acid. It was found that the patient could tolerate without insulin and glycosuria, a diet with a glucose content of 90 grams and composed of protein 53 grams, fat 210 grams and carbohydrate 37 grams.

Because of an infection (coryza) and a slight dietary indiscretion, the patient reentered the clinic in March, 1923, semicomatose, weighing 45 kilograms. After the infection had disappeared and the coma had been relieved, it was determined that he could still tolerate a diet with a total glucose content of 90 grams, composed of protein 53 grams, fat 210 grams, and carbohydrate 37 grams.

Because of undernutrition the patient was given, in March, 1923, a diet composed of protein 70 grams, fat 290 grams, and carbohydrate 75 grams, and a daily total of 26 (13-13) units of insulin in divided doses.

After five months of treatment with insulin, in August, 1923, it was found that he could continue to tolerate without glycosuria the same diet, which has a total glucose content of 90 grams. After an infection, coryza, in April, 1924, it was found necessary to take daily 46 units (16-15-15) of insulin in divided doses together with the same diet to remain aglycosuric.

In August, 1924, he found that with the same diet the daily insulin dosage could be reduced to 41 (21-10-10) units in divided doses, without a resulting glycosuria. It is believed that the reduction in dosage at this time was due to the slowness in the resolution of the infection (coryza) together with the increased muscular activity in the summer months. The diet remaining constant, the dosage was reduced to 40 (20-8-12) units daily in divided doses in December, 1924, without glycosuria. The same diet and insulin dosage were continued, and on December 31, 1924, a series of blood sugars were taken, with the following results:

TABLE I

Time	Insulin (units)	Blood Sugar %
7:00 A.M. (fasting)	..	.128
7:30 A.M.	20	...
8:00 A.M.	..	.153
11:30 A.M.	8	...
12:45 P.M.	..	.129
5:00 P.M.	12	...
6:00 P.M.	..	.073

In January, 1925, the diet was reduced to protein 66 grams, fat 255 grams, and carbohydrate 64 grams, with 42 (20-9-13) units of insulin per day. This dosage was reduced in June, 1925, to 34 (13-8-13) units daily, with the diet constant.

For the past one and one-half years the patient has been on this regime, aglycosuric and with only an occasional insulin reaction, which was induced by increased muscular activity such as tennis, swimming, canoeing, surveying on mountains.

In January, 1926, the patient, now a third year medical student, determined the amount of glucose in both his own and normal urines by the microscopical method of Shaffer and Hartman (10). Both the normal urines and his own, the diabetic, con-

tained from 400 to 900 milligrams of glucose per 24 hours.

In February, 1926, a fasting blood sugar was .136%. In August, 1926, because of hypoglycemic reactions after increased muscular activity, the daily dosage was reduced to 27 (13-5-9) units per day. The diet was unchanged. The patient now weighs 54.5 kilograms, takes the same diet and has reduced his dosage to 26 (12-5-9) units per day, without glycosuria.

Discussion

Case I was observed for nine months with a diabetic diet only, and for forty-two months with both diet and insulin. This case shows a gradually decreasing tolerance with a uniform diet and without insulin over a nine-month period. The gradual decrease from an original glucose tolerance of 90 grams, to 61, then to 46 grams was due to infection.

With a constant diet the dosage of the drug had to be increased from 9 to 59 units daily in two months time for the same reason—infection. On unchanged diet and dosage of insulin glycosuria and hypoglycemic reactions were avoided for 36 months. At the end of this period it was observed that three units of insulin could be omitted, so that the daily dose was 56 units. This was in part due to different spacing of the insulin dosage, but more especially perhaps to the omission of 5 grams of carbohydrate from the diet, which had been uniform for the past three years. Instead of insulin at 7:00 and 11:00 A.M. and 5:00 P.M., it was found that aglycosuria was only constantly present with the administration of the drug at 7:00 A.M., 5:00 and 10:00 P.M. Any further lowering of insulin or different time of dose caused glycosuria.

Case II shows that with diabetic diet alone the glucose tolerance decreased within four months from the original level of 71 grams to a number of grams somewhere between 64 and 50. He had insulin and was observed for 45 months. During the last three years the diet has been constant except with the small variance of from one to three grams of each of the food stuffs. To avoid glycosuria in the beginning, 28 units of insulin were necessary. Because of various infections the dosage had to be increased from 28 units to 36, then 48 and finally 85 units, with a constant diabetic diet.

It is exceedingly interesting and instructive to note that during the insulin dosage there are two long periods of time, during which an unchanged diet and insulin dosage both avoided

TABLE II

Case I. L. C.

Date	Diet P. Gms.	Diet F. Gms.	Diet CH Gms.	Total Glucose Gms.	Insulin Units	Urine Glucose
Nov., 1922	54	214	38	90	None	Neg.
Feb., 1923	31	176	28	61	None	Neg.
May, 1923	28	129	20	46	None	Neg.
June, 1923	55	220	35	88.9	9 (5-4)	Neg.
Dec., 1923	55	220	40	93.9	59 (22-14-23)	Neg.
Jan., 1926	55	220	35	88.9	56 (32-0-20-4)	Neg.

nyperinsulinemia and glycosuria. The first period of 20 months was from May, 1923, to January, 1925. The second of 12 months was between January, 1925, and January, 1926. The diet was absolutely constant throughout both of these periods, but the insulin dosage varied, being 36 units daily for the first period and 48 units for the second.

of insulin and a constant diabetic diet. for six months, the glucose tolerance when redetermined was 70 grams. The patient was observed for 32 months with daily insulin. It should be noted that although the diet was kept constant throughout this entire period, the original daily amount of insulin had to be increased gradually from 8 to 20 units, then to 30 units daily in order

TABLE III

Case II. R. B.

Date	Diet P. Gms.	Diet F. Gms.	Diet CH Gms.	Total Glucose Gms.	Insulin Units	Urine Glucose
Nov., 1922	40	180	30	71	None	Neg.
	28	129	20	50	None	Neg.
Mar., 1923	33	171	28	64	None	4 plus
Mar., 1923	58	281	57	118.7	28 (14-0-14)	Neg.
Apr., 1923	58	281	57	118.7	36 (18-0-18)	Neg.
Jan., 1925	58	281	57	118.7	48 (8-20-20)	Neg.
Jan., 1926	60	280	60	122.8	85 (25-30-30)	Neg.

TABLE IV

Case III. R. K.

Date	Diet P. Gms.	Diet F. Gms.	Diet CH Gms.	Total Glucose Gms.	Insulin Units	Urine Glucose
Mar., 1924	55	225	35	90	None	Neg.
Apr., 1924	55	230	45	99.9	8 (0-8-0)	Neg.
June, 1924	55	230	45	99.9	20 (0-20-0)	Neg.
Oct., 1924	55	230	45	99.9	30 (10-10-10)	Neg.
Mar., 1926	55	230	45	99.9	30 (10-10-10)	Neg.

Infection, influenza, between these periods had reduced the tolerance. It must be observed that the high dosage of 85 units daily and the same diet was the final amount given. The increased dosage was needed after coma with an acute upper respiratory infection.

Case III presents the most important evidence of any one of the five cases. The original glucose tolerance level was 90 grams. After daily doses

to eliminate glycosuria. The cause of each increase in dosage was glycosuria with infection. It is of interest to note that the patient has remained without glycosuria and hypoglycemia from October, 1924, to date on a constant diet and constant daily dosage of insulin. Any reduction in insulin or increase in the diet causes glycosuria.

Case IV shows the same end results with a constant daily insulin dosage for 36 months, but with a varied diet.

The insulin dosage has been 52 (32-20) units per day. The first diet with insulin contained a total glucose content of 126.9 grams, while the second had 116.8 grams. Although the insulin dosage was constant throughout, ten grams of the total glucose had to be removed from the first diet in order to keep the patient free from glycosuria.

found to be still 90 grams. For 21 months the same diet and daily dosage of insulin as shown in Table VI were given. Any reduction of insulin resulted in glycosuria. To maintain aglycosuria during infection, insulin dosage had to be increased to 46 units. As the infection gradually cleared, he was only able to reduce his daily dose to 40 units, with the same diet. His

TABLE V

Case IV. D. A. H.

Date	Diet P. Gms.	F. Gms.	CH Gms.	Total Glucose Gms.	Insulin Units	Urine Glucose
Nov., 1923	54	210	67	120	None	Neg.
Nov., 1923	55	250	70	126.9	52 (32-0-20)	Neg.
Apr., 1925	55	250	70	126.9	52 (32-0-20)	Neg.
Aug., 1925	60	220	60	116.8	52 (32-0-20)	Neg.
Apr., 1926	60	220	60	116.8	52 (32-0-20)	Neg.

TABLE VI

Case V. F. J.

Date	Diet P. Gms.	F. Gms.	CH Gms.	Total Glucose Gms.	Insulin Units	Urine Glucose
July, 1919	53	210	37	90	None	Neg.
Mar., 1923	53	210	37	90	None	Neg.
Mar., 1923	70	290	75	144.6	26 (13-13)	Neg.
Apr., 1924	70	290	75	144.6	46 (16-15-15)	Neg.
Aug., 1924	70	290	75	144.6	41 (21-10-10)	Neg.
Dec., 1924	70	290	75	144.6	40 (20-8-12)	Neg.
Jan., 1925	66	255	64	127.7	42 (20-9-13)	Neg.
June, 1925	66	255	64	127.7	34 (13-8-13)	Neg.
Aug., 1925	66	255	64	127.7	27 (13-5-9)	Neg.
Jan., 1927	66	255	64	127.7	26 (12-5-9)	Neg.

Case V is perhaps by far the most interesting and instructive. On a diabetic diet without insulin for 9 months, the glucose tolerance was the same at the beginning as at the end of the period, namely, 90 grams. This case was observed for 45 months with daily insulin. After he had received a constant diet and daily dose of insulin for 5 months, the glucose tolerance was

tolerance is now clearly less than at the beginning. In January, 1927, he was able to reduce the daily dosage to the original amount used in March, 1923, namely 26 units. The total glucose of the diet of March, 1923, was 144.6 grams, while that of January, 1927, was 127.7 grams. Although the insulin dosage remained uniform in order to avoid glycosuria, the diet of

January, 1927, must contain 16.9 grams less of glucose than did the diet of March, 1923.

CONCLUSIONS

To date there has been no evidence advanced which shows that the daily administration of insulin over long periods of time has been followed by a gain in the total glucose tolerance of a diabetic, which could not be explained by desugarization or recovery from infection.

A failure to obtain evidence of improvement in the tolerance for glucose in five patients, who had received weighed, high fat, low protein and low carbohydrate diets and daily insulin over periods varying from 32 to 45 months, has given us no ground for believing that insulin is capable of effecting a cure or a partial cure of human diabetes mellitus. Insulin has shown no more ability to arrest the downward progress of the disease than the earlier treatment without the drug. Each of these patients has lost tolerance during the period of treatment with insulin.

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The Conservative Treatment of Gangrene Complicating Diabetes*!

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THE treatment of gangrene complicating diabetes is a problem not strictly medical or surgical, but one which calls for the closest cooperation between surgeon and internist.

Often the type of treatment instituted depends on the specialty of the doctor first consulted. The general practitioner often considers gangrene a surgical problem and the surgeon usually sees the patients in consultation. The laity speaks of gangrene and amputation in the same sentence.

If surgery is the only method of treatment considered, limbs which might have been saved by conservative measures are amputated. On the contrary, if the internist fails to seek surgical counsel, continuing conservative treatment when amputation is indicated, toxemia or death may ensue; in either instance, an injustice is done the patient.

This paper deals principally with the medical phase of this problem and the measures available for conservative treatment.

*Read in Abstract before the California Medical Association, Los Angeles, California, April 28, 1927.

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HISTORY: Credit for the discovery of the relationship between glycosuria and gangrene probably should be given to Marchal (1) who, in 1852, presented a report to the Academy of Medicine in Paris. However, as early as 1806, Garco (2) pointed out that diabetics often suffered from ulcerations on the legs.

ETIOLOGY: There are many speculations relative to the cause of the vascular disease found in the majority of diabetic patients past middle age. Marchal (1) considered the possible effect of the long-continued hyperglycemia upon arteries. Joslin (3) has noted a positive Wassermann reaction in 11% of his diabetic patients suffering from gangrene, while the proportion was only 1.6% for 1,000 Wassermann tests in his series of diabetic patients. French writers (4, 5, 6) have also stressed the importance of syphilis as an etiological factor.

Letulle, Labbe, and Heitz (7) suggest that as a result of long-continued hypercolesterolemia, cholesterol may be retained in the intima of the arteries and thus promote calcification and obliteration of the lumens of the vessels. Their hypotheses are more or less confirmed by Labbe's (8) later report. He found a marked increase in the calcium content (10 times nor-

mal) in the sclerotic arterial walls of a diabetic sixty-two years of age. There was also a marked increase in the magnesium and cholesterin content. This patient's urine had shown sugar for eighteen years and he had symptoms of circulatory insufficiency in the legs for two years. Seven months prior to hospitalization, he developed an ulcer of the right fifth toe; four weeks prior to entry, two toes of the right foot had become black and very painful. The blood cholesterol was 2.50 on entrance (it fell to 1.85 eight days later, after the urine had become sugar free by the use of insulin). The blood calcium was .105 grams per litre. A month after entry, the right leg was amputated and the patient died a few hours later. The high blood cholesterol, followed by extension of the gangrene, and the death of this patient in a short time, confirms the ideas of Gray (9) in regard to the value of blood fat or cholesterol estimation in determining the prognosis in diabetes. Abnormal blood fat was found by this investigator in 78% of 1,062 specimens obtained from diabetic patients. He concluded from his tables that the higher the blood fat level (or cholesterol) the shorter the life expectancy.

The occurrence of vascular disease in Warren and Root's (10) series of cases was more closely related to the duration of the diabetes than to the patient's age at the time of onset of the symptoms.

Beard (11) has pointed out that exposure to cold may be a possible indirect cause of gangrene.

PATHOLOGY: The term "arteriosclerotic gangrene with diabetes" as proposed by Buerger (12) emphasizes the importance of arteriosclerosis as the essential lesion in the production of gangrene complicating diabetes. This author's (12) excellent book contains a detailed description of the pathological findings.

INCIDENCE: Joslin (3) states that gangrene is responsible for 1/5 of all diabetic deaths in Boston. Morrison (13) lists gangrene as a contributing cause of death in 23% of the fatal cases of diabetes in Boston between 1895 and 1913. Other investigators (14) report gangrene as a complication in 7% of 963 patients suffering from diabetes, admitted to the Peter Bent Brigham hospital between 1913 and 1925.

In this latter series (14) all but 4 patients were over fifty years of age. In Joslin's (3) series, when the onset of diabetes was between the ages of fifty-one and seventy, gangrene developed in 3.6% of the patients. In those who developed diabetes after the age of seventy, 1 in every 5 had gangrene. This same series records no complication of gangrene in patients under the age of thirty-one.

Joslin (3) states that 55 of his cases occurred in males and 26 in females. In Blotner and Fitz' (14) series there were 33 males and 22 females.

Diabetic gangrene usually originates in the lower extremities. In the last named series (14), the lower extremity was affected 65 times and the upper extremity 4 times. In Eliason and Wright's (15) series the great toe was affected in 28 out of 55 patients. Next in order were the fifth, fourth, third and second toes. This held true also

for arteriosclerotic gangrene without diabetes. Comparing the site of the original gangrene with ultimate fatalities, the percentage of death was greatest when the third toe was the original site. Next in order were the second, fourth, fifth and first toes.

Gangrene of other portions of the body, though rare, is reported (16, 17, 18, 19, 20, 21), as is its occurrence in juveniles (22).

SYMPTOMATOLOGY: A detailed description of the symptomatology of the various types of gangrene complicating diabetes will not be attempted. In the type primarily caused by arteriosclerosis, the appearance of a black spot or dark bleb may be preceded by burning, tingling or pain in the affected extremity and by intermittant lameness. There may be a history of trauma, such as stubbing the toe or stepping on a rock. In the infected type, the blackened infected area may follow the paring of a corn, a pin scratch, injury from a nail in the shoe or other trauma.

In the neuritic type, a painless perforating ulcer develops insidiously on the heel, the great toe, or under the distal end of the metatarsal. When seen early, arterial pulsations may be felt in the foot, but the Achilles and patellar reflexes usually are diminished or absent.

MEDICAL VERSUS SURGICAL TREATMENT: An analysis of the literature shows that when intensive conservative measures are employed as outlined by Stetten (23), McArthur (24) and Bernheim (25), results are very favorable.

Blotner and Fitz (14) state that except in some patients with superficial gangrene, there have been few cases which have healed spontaneously under medical care. Their medical efforts were confined to keeping blood sugar concentration and urine normal and treating the gangrenous part by heat, massage and other forms of physiotherapy.

Joslin (3) urges surgery at an early stage. He states that he has seen the bad rather than the favorable cases and this experience has probably led him to this decision.

Eliason and Wright (15) have recently analyzed 55 cases in which amputation was done for gangrene complicating diabetes and conclude that early high amputation is advisable in most cases.

SELECTION OF CASES FOR MEDICAL TREATMENT: Patients who show early signs of demarcation and sulcus formation separating the dead tissue from the living should be treated medically. Fulminating gangrene calls for immediate surgical attention. The decision as to the type of treatment in the borderline cases should only be made after careful consultation between surgeon and internist.

Each patient requires individual judgment. The following are the important points to be considered in deciding upon the safest and best method of procedure:

1. *History and Physical Findings*: The rapidity of the spread of the gangrene prior to hospitalization, the physical findings on entry, and the laboratory data regarding the diabetes or

other complicating conditions, are paramount factors in rendering a decision as to the type of treatment.

2. *Severity of the Diabetes*: If hyperglycemia is still present, with sugar in the urine, further dietary regime and insulin therapy may result in definite demarcation with regression of the gangrenous process. If the blood sugar is maintained at a fairly normal level with absence of sugar in the urine for many days and the gangrenous area progressively increases in size, surgery is indicated. If dietary regime, supplemented by insulin, fails to render the patient sugar free and the process is slightly but progressively advancing despite vigorous conservative means, the surgical consultant is justified in demanding that amputation be done without further delay.

3. *Degree of Vascular Occlusion*: The ultimate success or failure of treatment depends on the degree of patency of the vascular channels. Tests for circulatory sufficiency are of aid in determining the degree of obliteration of the vascular supply. Roentgenograms are very valuable in outlining arteriosclerosis. New methods of demonstrating the vascular tree to show the degree of thrombotic or atheromatous occlusion are described by Brooks (26, 27) and others (28, 29, 30). With each of these procedures exposure and temporary ligation of the main arterial trunk is necessary before the radio opaque substance can be injected. The ischemia produced by even temporary ligation is dangerous in the presence of gangrene, especially in those cases in which conservative measures are contemplated. Ligation of the main arterial supply is considered dangerous by Buerger and others (31). However, ligation of the femoral artery has been recently advised as a therapeutic measure (32).

The French (33) use the oscillometer of Pachon in estimating the arterial circulation in the limbs and consider it the best available method for determining the degree and location of obliteration.

The intracutaneous salt test (34) is simple and safe and may be done on every patient. It is stated that the disappearance of the wheal in from 10 to 25 minutes is strongly suggestive of developing gangrene.

4. *Extent of the Gangrene*: It is difficult to formulate any set rules for guidance regarding the extent of the gangrene in selecting cases for treatment. There are many contingencies such as infection, extension of the process, location and degree of vascular occlusion modifying any general principles one may formulate. The prognosis in non-infected gangrene is of course better than in the infected type.

The following rules are applicable in determining conservative treatment in the infected type: (a) If the gangrenous process is limited to the digits, even though the phalanx shows necrosis, the chances for demarcation and spontaneous amputation with cure, are excellent. (b) If the gangrenous process involves the foot without death of more than 1/8 of the soft tissues and without signs of necrosis of bones other than the phalanges, the prognosis is fair. (c) If 1/8 to 1/4 of the dor-

sum or plantar surface of the foot is dead, or if the gangrene of the foot is complicated by necrosis of the bones of the foot other than the distal tip of the metatarsals and phalanges, conservative measures probably will not be successful. Roentgenograms are valuable in determining the amount of bone necrosis.

5. *Pain*: This is usually not so severe in the diabetic type of gangrene as it is in other types. Contrast baths may be of benefit; in fact some patients discover this method of relief before consulting a physician. Usually the pain is alleviated after the patient has had the advantage of the measures advised to enhance the circulation of the affected extremities.

6. *The Economic Aspect*: It is unfortunate that the economic situation should be an important factor in rendering a decision. However, if conservative treatment is not available at a low cost, amputation may be necessary for those patients who are unable to afford long hospitalization.

7. *Cooperation*: Before beginning treatment it should be explained to the patient that considerable time is required to secure results and that his strict cooperation will be necessary. If the individual's personality suggests that his cooperation will not be obtainable, he should be treated surgically.

CONSERVATIVE TREATMENT:

1. *Diet and Insulin* are directed along the same lines as for any diabetic patient over fifty years of age. The diabetes is usually of long standing and mild. However, the presence of infection may seriously lower the carbohydrate tolerance. The caloric intake should be regulated to maintain optimum nutrition. The patient should not be allowed to become overweight. The findings of Letulle (7) and others lead us to conclude that perhaps a high fat diet contributes to the production of arteriosclerosis. Since we are interested in arteriosclerosis as well as diabetes as an etiological factor and problem in treatment of diabetic gangrene, a low fat diet may be advisable as a prophylactic measure.

2. *Rest in Bed*: Activity calls for increased blood supply which the atheromatous and occluded arteries are unable to furnish. At rest, nutritional demands are minimal and the supply may be sufficient to carry on until a more generous collateral circulation is established. Simple exercises should be carried out daily. The use of "blow-bottles" may be valuable in preventing respiratory complications.

3. *Position*: As pointed out by Buerger (12), the level at which the color of the affected part is nearest normal should serve as the patient's horizontal resting position. In the presence of gangrene this position of maximum circulatory activity is usually below the horizontal plane. The bed should be so arranged as to maintain this position at all times.

4. *Postural Treatment*: Buerger's (12) postural treatment is contraindicated in the presence of gangrene. It is not only difficult to carry out, but may cause severe pain. The ischemic phase of this therapeutic measure is dangerous when the blood supply to the extremities is minimal. With fre-

quent change in position, there is greater liability to injury, with serious consequences.

5. *Fluids*: In 1912, Mayesima (35) reported an increased blood viscosity in thrombo-angiitis obliterans with gangrene. He also noted that the blood viscosity could be decreased by infusions of normal salt solution with favorable effect on the gangrene. The following year, Koga (36) confirmed these findings and reported a series of 13 patients successfully treated by infusions of normal salt or Ringer's solution. In 1919, McArthur (24) used this same form of treatment in diabetic gangrene and reported favorable results in 9 patients. In addition to giving Ringer's solution by hypodermoclysis and intravenously, he introduced 3 to 4 litres of this solution into the duodenum by means of the Rehfuss tube. In most of the patients the extent of the gangrene was sufficient to have justified amputation. McArthur (24) stressed the importance of trying this conservative measure before resorting to surgery.

When treatment is first instituted, it is advisable to give as much fluid by hypodermoclysis as the patient is able to absorb, in addition to forcing fluids by mouth. At the end of a week, the patient can usually be persuaded to drink 6 to 10 litres of fluid in lieu of the hypodermoclysis. In some cases it may be advisable to give 200-300 cc daily of normal saline intravenously. In our experience, normal salt solution hypodermically and intravenously and water by mouth have been just as efficacious as Ringer's, Locke's, sodium citrate, or other solutions. The beneficial effects probably depend upon the administration of fluid per se, rather than to the type of fluid administered. Owing to its greater penetration into the tissues, fluid by hypodermoclysis is possibly more valuable than an equal quantity taken by mouth. The daily intake should be at least 6 litres—the larger the amount, the more beneficial will be the effect.

There are no satisfactory scientific data to explain the beneficial effects of large quantities of fluid in the treatment of gangrene. Mayesima (35) advanced the theory that hypodermoclysis of normal salt solution favorably influences the progress of gangrene by decreasing the blood viscosity. Further studies are needed to confirm or disprove this theory. Stern lists one patient with diabetic gangrene and several other patients with various types of gangrene who showed normal blood viscosities.

6. *Thermotherapy*: The efficacy of superheated air has been pointed out by Meyer (37) and Stetten (23). Dieulefoy (38) has recommended its use at temperatures of 200 to 600 degrees, C., in exposures of one-half hour to three hours twice a day over the gangrenous area to prevent the spread of the process and hasten mummification. Stetten (23) has inserted the entire wrapped limb into a suitable oven and gradually worked up to temperatures of 180 to 200 degrees F. The maximum treatment advised was one-half hour twice a day if the patients were able to stand it. These treatments were followed by soaks in 100 degrees F. physiological salt solution for a half hour, immersing the entire limb.

This technique is difficult to carry out and exposure to high temperatures is dangerous, especially if the gangrenous area is infected. If the affected limb is kept constantly warm by a temperature of 100 to 125 degrees, F., better results are obtained. This may be accomplished by means of a carbon filament lamp, with reflector, attached to the roof of a cradle which has been covered with two or three thicknesses of blanket. A wire screen or guard over the light prevents any portion of the body from coming into direct contact with it. The apparatus is placed over the affected extremity and a switch placed conveniently at the head of the bed. The light is operated by the patient who soon becomes expert in maintaining the proper temperature. Treatment should be continuous during the waking hours. A constant vasodilation by reflex action is thus maintained. There have been no untoward effects or accentuation of symptoms from this treatment.

Hot compresses of 1-1000 neutral acriflavin or gentian violet are applied continuously to the affected part as long as infection is present. Heat is maintained in the compresses by the hot air apparatus. When the odor is particularly offensive, hot compresses of ¼% phenol solution are used. This causes very little, if any, devitalizing effect on the tissues. When infection is no longer present, compresses may be discontinued. In this stage exposure to sunlight or the Quartz Mercury Lamp may aid granulation.

7. *Diathermy*: Theoretically, diathermy should be of infinite value as it warms the deeper tissues and produces vaso-dilation of the smaller arterioles and capillaries. In 1915, Wolf (39) treated a diabetic woman seventy-eight years old, who suffered excruciating pain from an arteriosclerotic gangrenous ulcer on the plantar side of the little toe. By the use of diathermy, the ulcer healed in four weeks and did not reappear. Cluzet and Chevallier (40) report five cases of moist gangrene of the leg and one case of moist gangrene of the scrotum, completely cured by this form of treatment. Improvement was noted from the first treatment and the favorable effect on the circulation was shown by the change in the oscillometer record. Insulin seemed unnecessary for recovery. These authors believe diathermy may save similarly affected limbs from amputation. Lian and Descoust (41) are also enthusiastic supporters of diathermy and believe that it produces a local and general warming effect and intense vaso-dilation which assists the flow of blood into the smaller arterioles. In addition, they say that diathermy combats the spasms which are often concomitant and exercises an eutrophic action on the tissues, increasing their metabolism and improving nutrition. The results of other investigators have not been so encouraging. Buerger (12) says that diathermy is not well borne nor beneficial in the presence of inflammation, ulceration or gangrene.

Heat, however, applied, is of extreme importance in helping to re-establish circulation.

8. *Massage*: This form of therapy is considered inadvisable by some authors on account of the possibility of detaching thrombotic occlusions in

the vessels. The advantages of light effleurage are greater than the danger of embolism. It serves to maintain the normal tissue turgor and elimination from the skin. From the work of Boas (42), one may assume that reflex stimulation by massage may either cause relaxation of contracted capillaries or may increase the tone of the toxically paralysed smaller vessels whose lumens are obliterated by collapse of their walls.

9. *Medication*: The French (43, 44, 45) believe that sodium citrate by mouth is of definite value in the treatment of the various forms of gangrene. Labbe (4) states that the use of 20 to 30 grams daily may give good results by calming the pain and aiding the circulation. Our experience confirms these findings. Perhaps the beneficial effects may be explained by stimulation of the muscular tonicity of the vessels innervated by the sympathetic system.

Renaud (46) reports a gradual increase of both systolic and diastolic arterial tension following the injection of 8 to 12 grams of sodium citrate over a period of time. Four of our patients were given 12 to 16 grams of sodium citrate by mouth for a period of eight months without any increase in systolic or diastolic arterial tension, in fact both determinations were lower than before the administration of sodium citrate.

Mayesima (35) believed that the internal use of potassium iodide by those suffering from gangrene exerted a beneficial effect by decreasing the blood viscosity. These observations have not been confirmed. It may be of value in promoting the absorption of cellular exudates and should be included in the patient's medication. It is prescribed in 2 cc. doses of the saturated solution three times a day.

If pain is severe, pyramidon, atropin or a combination of acetyl-salicylic acid and morphine may be tried for relief. Vasodilators are usually of no benefit.

If syphilis is suspected despite negative tests, approved anti-syphilitic treatment should be given. In these cases mercury ointment may be massaged into the skin overlying the main vessels of the extremities. Anti-syphilitic treatment is particularly efficacious in the neuritic type of gangrene.

Thyroid extract to the point of toxicity may be prescribed, especially if the basal metabolic rate is low. Physiologically it is said to produce dilatation of the peripheral vessels.

The possibility of tetanus (47) and gas gangrene must also be kept in mind. It may be advisable to give periodic prophylactic doses of tetanus antitoxin.

10. *Vaccine and Protein Shock*: Singer (48) has used intramuscular injections of caseosan with very favorable results in three cases of extensive diabetic gangrene. Pchellas (49) reports success in one case, by the use of mixed infection phylacogen.

11. *Drainage*: Infected moist gangrene is the type usually seen complicating diabetes. Proper drainage should be instituted early and the patient's progress followed by the surgeon. The area should be inspected and probed daily to open up any suspicious areas in the wound.

12. *Occupational Therapy*: Even with a satisfactory condition of the affected extremity a problem still remains in the preservation of the patient's morale. Basket weaving, tapestry or rug making and other forms of handicraft may be taught and some patients become very adept and enthusiastic workers. Sale of these products may help solve the financial problem.

COURSE IN THE HOSPITAL: If progress is favorable, the sulcus of demarcation gradually becomes wider, exposing the pale red cross section of skin on the healthy side. The black skin covering the affected portion becomes progressively dryer and contracts, exposing the white, necrotic grayish-pus soaked deeper tissues. The under surface of the skin covering the viable area presents the same appearance. The phalanx, if exposed, is soft and spongy. Sooner or later, spontaneous amputation occurs. Visualize strands of fascia and tendons with mushy shreds of muscle and protruding necrotic bone with a grayish-black offensive purulent discharge and you have a fairly accurate mental picture of the progress so far.

The discharge becomes progressively less and the wound looks cleaner. Long strands of dead tendon and fascia may now be snipped off, but no attempt should be made to pull these strands out from the wound or pull on them so that they may be cut off as short as possible as this may result in reinfection and loss of tissue above the line of demarcation.

The picture now is much more encouraging and the patient is happy because he can see favorable progress. As the wound is probed for small collections of pus, blood oozes freely from the solid, red, healthy granulations. The threads of diseased tissue and the remaining parts of the bone slough off and healthy tissue fills up the site. Epidermatization is followed by discharge from daily observation.

The usual duration of treatment is from two to six months, depending on the severity and extent of the gangrene and the degree of vascular occlusion.

DISCHARGE INSTRUCTIONS: At the time of discharge from the hospital, a written detailed list of instructions should be given to the patient. This is of supreme importance. Joslin (3) stresses the necessity of "keeping the feet as clean as the face." The patient should be warned against all factors such as prolonged standing, long walks, tight shoes, elastic bands, etc., which tend to disturb the fine balance between arterial sufficiency with tissue preservation and arterial insufficiency with trophic changes. Any tissue damage, such as abrasions, scratches, blisters or burns, however trivial, should be given immediate and careful attention. Before cutting nails the feet should be thoroughly cleansed and bathed in alcohol and all soft tissue injury should be avoided. Long nails, or sharp points left after cutting may produce injury leading to serious consequences. Corns, bunions and ingrown nails should be treated by a surgeon. In the event of any infection the patient should report immediately. Hose of suitable thickness should be worn so that the feet are kept comfortably warm. When the

patient is at leisure, the shoes should be removed and the feet elevated to the angle of circulatory sufficiency.

Tobacco and alcohol are prohibited. The daily fluid intake should be from 3,000 to 4,000 cc. The blood sugar concentration and urine should be kept normal by suitable diet and the use of insulin. The patient's weight should not exceed normal. An apparatus such as described for maintaining hyperemia can easily be made by the patient for use in his home. After discharge from the hospital, the extremities should be placed in this hot air apparatus for at least three hours daily. In some cases a longer exposure may be advisable.

Instruction regarding Buerger's (12) postural treatment should be given and one to three seances daily of this form of therapy may be very valuable as a prophylactic measure. Sodium citrate in 2 dram doses three times a day should be continued as well as the potassium iodide. The patient is, of course, instructed to report immediately if sugar occurs in the urine. Periodic fasting blood sugar determinations are made once a month or oftener.

The value of prophylactic treatment should be reiterated each time the patient is seen. The gospel of prevention should be impressed upon every diabetic, particularly those over fifty years of age. If he is inclined to belittle the possibility of gangrene as applied to himself, he may be impressed by a visit to the hospital to see the effects, on other patients, of neglect of prophylactic measures.

The liability to recurrence is the greatest hazard following discharge from immediate supervision. If the patient conforms strictly to the instructions given on discharge, the probability of recurrence is minimized. If a comprehensive course of treatment directed toward maintaining circulatory sufficiency has been given, a recurrence of gangrene following trauma or infection usually clears up after a short stay in the hospital on the regime advised. This is exemplified by the following case reports:

Mrs. A. H., age 69, July 6, 1925. Duration of diabetes 12 or 13 years. Local trouble began one week before entry, following stepping on a rock. Chronic ulcer at the plantar base of the second left toe, for 4 months.

Examination: Phlegmon at the anterior base of the second left toe, ulcer about the size of a dime at the plantar base of the second left toe, with discharging sinus, area of infected moist gangrene about the size of a dollar on the mid-plantar surface of the left foot; foot red and edematous. Fever 100. Marked arteriosclerosis. No pulsations felt in the foot. Popliteal pulse feeble. General condition fair. Blood sugar 285.7 mgm. per 100 cc. of blood. Blood Wassermann negative. Urinalysis: sugar 3.8%, diacetic marked, acetone moderate; albumin trace; occasional hyaline and granular casts.

Routine conservative treatment, with incision and through and through drainage of the ulcerated and gangrenous areas.

November 2, 1925: Foot almost healed. The patient's condition satisfactory for discharge, but she preferred to remain until the foot was entirely healed.

December 24, 1925: Patient discharged. Foot completely healed.

March 9, 1926: Patient re-entered the hospital: Had stubbed left foot three days before entry.

Examination: Gangrenous blebs on the tip of the great toe, the tip and side of the little toe and several blebs along the lateral side of the left foot. Blood sugar 190 mgm.

per 100 cc. of blood. Urinalysis: sugar negative, diacetic and acetone negative.

April 15, 1926: Patient discharged. Foot healed. No recurrence to date.

Mrs. E. B., age 69, May 2, 1926. Duration of the diabetes unknown, estimated about 7 years. Three months before entry, infection of the right great toe followed the application of a corn plaster. The area had been discharging for two weeks.

Examination: Moist infected gangrene of entire right great toe. Foot swollen and edematous. Blood sugar 377 mgm. per 100 cc. of blood. Blood Wassermann negative. Blood plasma CO-2, 38.5 cc. bound as bicarbonate by 100 cc. plasma. Urinalysis: sugar 4.8%, acetone and diacetic negative, albumin trace. Routine conservative treatment instituted.

June 12, 1826: Toe sloughed off.

July 18, 1926: Patient discharged; foot healed except for a very small denuded area. September 29, 1926: Re-entered hospital. On September 16, ran a small nail into the mid medial surface of arch of right foot with subsequent infection of this area and the side of the foot. Sugar found in the urine for 4 days before entry.

Examination: Gangrenous phlegmon of the arch of the right foot. No sugar in the urine.

Incision of the area with drainage.

October 13, 1926: Patient discharged with a slight drainage from the site of incision but in a satisfactory condition. No recurrence to date.

In the past, attention has been directed principally to the dietary treatment of diabetes and the surgical treatment of gangrene. However, there have been reports (50) of successful conservative treatment of diabetic gangrene antedating the advent of our modern treatment of diabetes. Since the discovery of insulin and the newer methods of dietary administration, the treatment of diabetes has been revolutionized.

DuPre (51) and others (52, 53) have reported remarkable results in the conservative management of gangrene by the use of diet and insulin alone.

Insulin is an invaluable aid in quickly controlling diabetes even in cases of infected moist gangrene, thereby improving the patient's nutrition and increasing his resistance. By this indirect effect it is of inestimable value in the treatment of diabetic gangrene. This is shown by the figures of Blotner and Fitz (14):

Of 13 cases treated medically without insulin ..... 3 died

Of 11 cases treated medically with insulin ..... 2 died

Of 40 cases treated surgically without insulin ..... 10 died

Of 9 cases treated surgically with insulin ..... 1 died

Of 53 cases, medical and surgical, treated without insulin, 13, 30% died

Of 20 cases, medical and surgical, treated with insulin, 3, 15% died

Infection, although still a very potent factor, is not so serious in dealing with diabetes as it was before the use of insulin. With the application of ordinary surgical principles, ulcers, abscesses and other infections, including osteomyelitis, rapidly clear up, after the urine has become sugar free by the aid of insulin.

Time and expense are the principal objections to medical treatment. Unless these objections are insuperable, the patient is more than compensated for the expenditures by the preservation of his extremities.

Results from surgical treatment of diabetic gangrene are not particularly encouraging. Joslin (3) says that of 43 of his patients treated surgically, 15 or 35% died during the first year. Eliason and Wright (15) give their mortality as 43.6% in a series of 55 cases.

When amputation is necessary, it should not be the sole method of treatment. Following operation all patients should receive the benefits of the treatment outlined for increasing the circulation of the extremities. The advantages are noticeable in the rapidity of healing of the stump. Most surgeons do not consider this phase of the treatment of diabetic gangrene, and fail to give the patient adequate instructions relative to prophylaxis on their discharge from the hospital.

In advanced cases in which amputation is definitely indicated, but refused, remarkable results may be obtained and the patient's life saved by adequate conservative treatment.

If patients suffering from diabetic gangrene are given the advantage of the measures outlined for enhancing the circulation in addition to approved treatment for their diabetes, many favorable reports from conservative treatment will be recorded in the literature in the future.

Try conservative treatment in selected cases of diabetic gangrene, but do not let your enthusiasm replace sound judgment. Be conservative as long as progress is satisfactory, but be open to conviction if surgical counsel doubts favorable progress and suggests possible beginning toxemia.

CONCLUSIONS

1. Selected cases of diabetic gangrene including the moist infected type may be successfully treated by conservative measures.
2. Gangrene of sufficient extent to ordinarily justify amputation of the extremity may be treated conservatively and the extremity saved. Many of these gangrenous extremities which might have been saved by conservative measures have been sacrificed.
3. Too little attention has been paid to conservative measures which enhance the circulation of the gangrenous extremity. When treated surgically, the patient should have the advantage of these measures before and after amputation.
4. Insulin is of inestimable value in the treatment of diabetic gangrene in that it quickly controls the diabetes, thereby improving the patient's nutrition and increasing his resistance to infection.
5. Prophylactic treatment should include all measures which improve the circulation of the extremities.
6. If treatment is continuously employed to improve the circulation of the extremities the possibility of recurrence of gangrene is minimized.
7. If the patient has had a comprehensive course of treatment directed toward maintaining circulatory sufficiency, recurrence of gangrene following trauma or infection usually clears up after a short stay in the hospital.

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The Relation of Trauma to Cardiac Disease

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THE medico-legal phase of the problem whether indirect injury to the thorax can induce temporary or permanent damage to the myocardium is sufficiently important to justify the addition of one other case report of this type. The medical expert is frequently in grave doubt whether a myocardial involvement or a disturbance of rhythm should be ascribed to a previous injury or trauma. Included in this category is the matter of heat exhaustion or heat stroke as a factor in the production of permanent myocardial involvement with a disturbance of rhythm. The matter is of considerable interest to clinicians aside from its very practical importance in a legal way.

Bockbank, in his book on "Incapacity and Disablement in its Medical Aspects," states that he has not seen evidence that auricular fibrillation could be brought on by sudden strain and that he is unwilling to believe that such strain could produce it. He does not make clear in this opinion whether he refers to strain superimposed upon damaged hearts, or whether he has reference only to hearts not previously involved.

The opinion that auricular fibrillation occurs only in hearts which have been damaged by existing pathological changes of whatever nature, undoubtedly holds true in the great majority of cases. The fact that such pathological change had not previously been demonstrated does not invalidate this opinion. The medico-legal bearing of this problem has to do also with the production of cardiac arhythmias and their resultant conditions in hearts which have actually shown pre-existing damage. The law does not hold that an employer is free from responsibility, even though the heart were not entirely normal prior to the injury or strain. The role of the injury in bringing about the cardiac condition as determined at the existing period is a question to be determined at the legal investigation.

The second problem to be considered in this consideration is the possible effect of a temporary auricular fibrillation in its tendency to recur or become permanent. The fact that auricular fibrillation tends to recur in cardiac states even after long periods of absence is well known. Whether a heart which has undergone fibrillation following injury will have this same tendency to recur or become permanent is an unsettled problem. The presence of auricular fibrillation is undoubted reason for damage claims for disability. Also, the future probability of an individual to have a recurrence is a very practical consideration in the

assessment of damages or the placing of responsibility.

It is hardly necessary at this point to list the symptoms of auricular fibrillation as impediments to an employee. The increased shortness of breath, the cardiac or thoracic distress, the mental and nervous symptoms, are all well known and grave hindrances to normal productivity on the part of individuals engaged in any occupation.

Eyster and Swarthout estimate that auricular fibrillation in dogs decreases the output of the heart by 40 per cent, even when the myocardium is normal and the irregularity has been brought on by experimental means. This very considerable decrease in the cardiac output indicates how poor and inadequate is the circulation in this form of arhythmia. This decrease in flow from the heart necessitates the great increase in the rate which often reaches figures of 140, 150, or 160.

Every physician has had the experience of meeting in his practice individuals showing auricular fibrillation who have not been aware of such disability. Such individuals may not have complained of cardiac symptoms, but a careful analysis of their history reveals a definite slowing down of their activities and a disinclination to prolonged or arduous labor and strain. There is a second group in which the fibrillation follows definitely a particular cause, with or without immediate recognition of the time of its onset. Such predisposing causes are numerous, including thyroid states, infectious diseases, and other well known conditions.

Hay and Jones (1), who have recently written upon this subject, listed instances of auricular fibrillation following, respectively, exposure to electric current, sudden physical effort and alarm, following a struggle, and onset after sudden effort and lifting heavy weights.

Gossage and Hicks (2) reported several cases in 1913 in which the fibrillation occurred without apparent reason in young patients with apparently healthy hearts. This possibility does not seem so likely to occur at the present time with our greater opportunities for recognizing obscure thyroid states and other infectious processes.

The instance I wish to record is as follows:

A. G. Age 20. Occupation, truck driver. This man was admitted to St. Vincent's Hospital April 24, 1927, with the history that while engaged in some repair work on his truck train, he was caught between two trucks and rather severely crushed in the thoracic region. He was not rendered unconscious and there were no fractures determined by the x-ray. There were no external wounds, cuts, or lacerations, when he was seen shortly after his injury. Later, a few discolorations appeared from bruising. However, there was no question that he sustained a very severe crushing injury by two trucks which were forced together with him between.

The patient complained of severe pain in the thoracic region generally and also of marked distress in the cardiac region and breathlessness. He was not cyanotic. Examination showed very definite auricular fibrillation. The heart action showed complete irregularity when he was first

seen by me. The heart rate was counted at 150. The pulse was 60, but this could not be determined with absolute accuracy. There was considerable tenderness over the chest region generally. The pupils of both eyes were considerably dilated. The patient was weak, but clearly conscious and able to speak without any trouble. Deep breathing was difficult on account of the painful movement of the respiratory muscles. This patient was seen also by Dr. Thomas Crinnion and Dr. Frank Clifford. The auricular fibrillation persisted for 24 hours. It is regrettable that an electrocardiogram was not secured during these first few hours, but he was too sick to be moved. Any movement caused him much pain and greatly increased his breathlessness. However, it semed to me that there was no question whatever that there was an absolute irregularity of the heart action, that is, an auricular fibrillation. The increased rate and the great pulse deficit further supported this diagnosis.

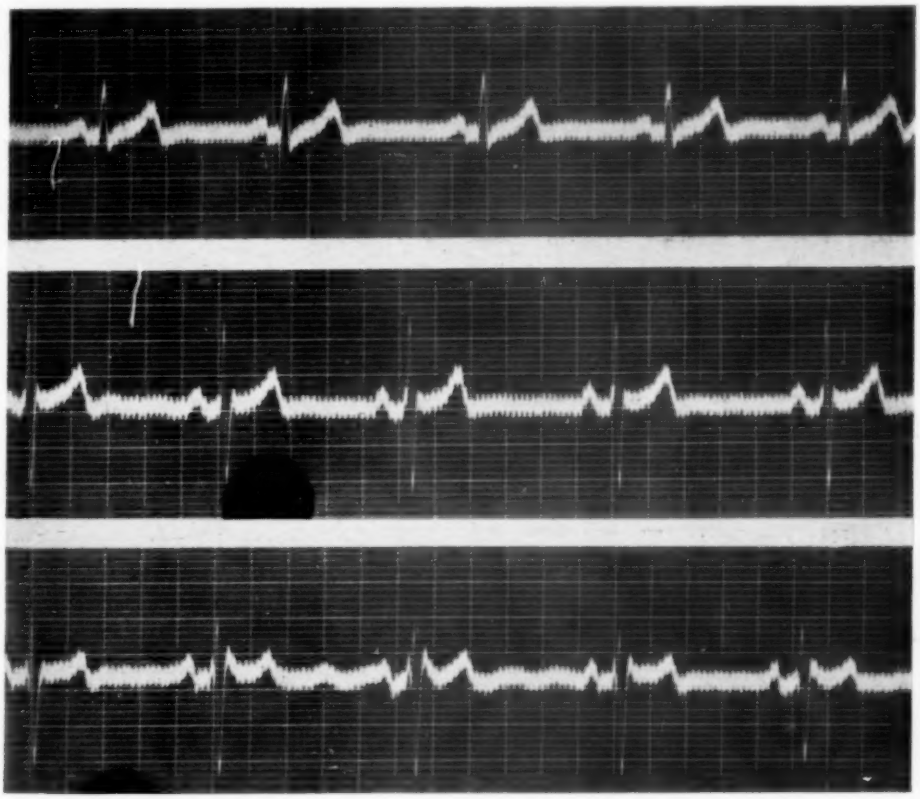

FIG. 1. Electrocardiogram 36 hours after injury.

FIG. I

An electrocardiogram was taken 36 hours after the injury. (Fig. 1.) This examination showed the 'P-R' interval

0.18, the Q-R-S complex 0.08 in duration. The T waves in leads I, II, and III were abnormally large. There was definite left ventricular preponderance. The rhythm showed very moderate sinus arhythmia. The heart action seemed to be normal in a clinical way at the time this electrocardiogram was obtained. The patient complained of considerable pain in the region of his thorax and back, but the sense of cardiac distress and breathlessness was absent. The increased heart rate had returned to normal and the pulse deficit was no longer present. Blood pressure was 120/82.

Later, when this patient had apparently recovered, he was subjected to a very careful examination and analysis of his previous life, working conditions, and possible symptoms. It was brought out very definitely that he had always been well and as far as he knew had never had any symptoms which could possible be connected with his heart or which impaired his working capacity. Clinical examination of the heart later has not revealed any evidence of cardiac disease in any way. The heart and blood pressure are apparently entirely normal. Physical examination generally did not reveal any thyroid disease, toxic state, or infection, which might have brought on a transient period of fibrillation. It is my impression that the fibrillation in this case was brought on by the crushing injury to his thorax. The question whether there will ever be a tendency to a recurrence of this form of cardiac arhythmia in his case can not be answered at this time.

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Basal Blood Pressure in the Normal*

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PROBABLY one of the greatest advances in internal medicine during the last half century has been the increasingly more common recognition of the fact that the important consideration in evaluating the condition of the heart is its capacity for doing work. Today the detection of murmurs is only a small part—and often an insignificant part—of the problem of estimating the health of the heart.

Similarly the mere determination of a blood pressure of a given number of millimeters falls far short of giving us a definite prognosis of any case. Ehrstrom (1) in analyzing a group of 20 who were observed ten to sixteen years with a blood pressure constantly (or nearly so) of two hundred or more found that they felt and looked well and continued to work. From a study of 300 cases he comes to the conclusion that the chances are about even as to whether a high blood pressure means a shortening of life. Even in hypertensive patients with heart disease he found unexpected remissions or long tolerance.

Because of this wide variation in significance of any blood pressure finding even with the utmost care in the selection and manipulation of the instrument and the proper control of the individual, numerous attempts have been made to develop tests which would extend the value of blood pressure determinations.

One of the first questions to be answered by the physician who finds a pressure of say two hundred is whether or not that pressure is temporary and will soon pass away or whether it tends to remain at that level and will not only not drop spontaneously but will be resistant to treatment. Of course a due consideration of all the data from a complete physical examination is necessary in the interpretation of any blood pressure finding, but this study at the best gives only indefinite information.

One of the most recent attempts to find a more definite answer to the question of the significance of a high blood pressure determination is reported by Sladen and Johnston (2). The blood pressure is taken under conditions somewhat similar to those required for the determination of the basal metabolic rate. This is called the basal blood pressure and very often it is found to be much lower than a reading obtained on first examination. Immediately afterwards a simple exercise is given and the systolic blood

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pressure taken at one-half minute intervals following the exercise and a graph plotted of all the determinations. According to these workers, if the curve rises immediately after the exercise and then falls sharply, reaching a level considerably below that basal rate in a few minutes, the prognosis is most favorable. On the other hand if there is only a slight and gradual fall and the basal rate is not reached, then there is little hope of effecting any considerable decrease in the blood pressure of that individual by any method of treatment and the patient must adjust his life permanently in accordance with the limitations set by that pressure.

Unfortunately only three cases are reported on which this test was made. Because of the simplicity of the test and because of the importance of the problem involved, it was thought advisable to investigate the test further and to begin doing this on the apparently normal individual. It should be pointed out that the following data are not presented as being contributions to the large problem of the effect of exercise on blood pressure, especially since it is realized as pointed out by MacWilliam (3) that it is necessary to measure the pressure during exercise if the complete picture of the changes is to be obtained. Rather was this study instituted as a beginning to ascertain whether the apparently normal individual reacted with any degree of constancy to this particular test.

The subjects chosen were university students of both sexes who volunteered for the test. They were in all cases in excellent health and were in rather better than average physical condition, being for the most part majors in the school of physical education. Not only were they quite used to having blood pressure taken but they also had this test described to them in detail. At the time an appointment was made they were given a mimeographed statement of what was expected of them with an explanation of just what was involved in the test so that they not only knew what to do but were free from any anxiety. To get their pressure at more nearly basal conditions they were asked to sleep in the University Infirmary and the test was made early the next morning before breakfast.

There were 28 men and 32 women tested. All of these had previously been found free of major defects and had had at least one normal urinalysis. A mercury manometer was used and all readings were taken with the patient recumbent—the cuff on the left arm. The exercise consisted in running up and down a flight of seventeen steps. For the safe of uniformity the men were all examined by the same physician as were the women. About half had their pulse rate recorded by an attendant, and in every case the pulse rate returned to approximately the rate before exercise in at least three minutes, and usually had returned to its original rate in one minute. The pressure was taken as soon after exercise as possible and thereafter at minute or half-minute intervals for about ten minutes. Both the systolic and diastolic pressure were recorded and the pulse pressure computed. Inasmuch as this work was undertaken as a study of the test advocated by Sladen and Johnston (1) in

which only systolic pressure is considered, it is only of our systolic pressure determinations that a detailed analysis is given in this report.

The most common type of reaction was an initial high rise followed by a sharp drop and then a very gradual drop to below the basal rate. This is the type of curve that Sladen and Johnston (1) consider of good prognosis. However, while this reaction is the most common, there is a high percentage of variations from it in the apparently normal. Tables 1 and 2 show in detail some of these variations. From Table 1 it will be seen that about 18% of those examined gave a systolic reading immediately after exercise that was not appreciably higher than the basal rate and in about 7% was actually less. In all cases there was a fall from this reading taken immediately after exercise, but the time this fall continued varied from one to fourteen minutes. In 40% of those examined the systolic pressure did not fall below the original or basal rate. Probably if a more involved type of exercise had been used so that measurements could have been taken during exercise a more uniform type of curve would have been obtained.

An analysis of the diastolic pressures gave no more uniform results, for in 43% there was no change (or less than 4 mm. change) after this exercise test described above—in 17% there was a rise and in 40% there was a fall. The most constant finding was a rise in pulse pressure immediately after exercise which occurred in 52 or 87%. No appreciable change (4 mm. plus) occurred in 13%. There was never an initial fall in pulse pressure. This was the only constant finding.

TABLE 1. Primary Change.

Changes from basal blood pressure (systolic) rate immediately after a given exercise.

Amount of change	Number of Men	Number of Women	Total	Percentage
Decrease of 4 mm. or more	2	2	4	6.7
No change	0	1	1	1.7
Increase of 1 - 3 mm.	3	0	3	5.0
" " 4 - 6 mm.	2	1	3	5.0
" " 7 - 12 mm.	6	7	13	21.7
" " 13 - 20 mm.	9	7	16	26.7
" " 21 mm. plus	6	14	20	33.3

TABLE 2. Secondary Change.

Change in pressure during rest period after exercise.

	Number of Men	Number of Women	Total	Percentage
No. dropped at least 4 mm. below basal rate	23	13	36	60
No. dropped to basal rate (or within 4 mm.)	4	13	17	28.3
No. that did not drop to within 4 mm. of basal rate	1	6	7	11.7

Conclusion. The variations in the systolic (and diastolic) pressure response noted after a simple exercise test in 60 normal young adults were so great that it seems extremely doubtful whether the test can be of any clinical value at least in its present form. There was a marked variation in (1) the initial change, (2) the rate of fall and (3) the amount of the fall.

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Torula Infection in Man

By Oscar Berghausen, B.A., M.D., *Cincinnati, Ohio*

ALTHOUGH medical literature records only a small number of cases due to infection with the yeast organism, *Torula histolytica*, it is assumed that many cases do occur but remain unrecognized. In 1926 Rappaport and Kaplan (1) reported the thirteenth case, the first one in which a lesion of the skin was described; Lynch and Rose (2) reported the seventeenth case, the infection involving the central nervous system; McGehee and Michelson (3) reported the isolation of the organism from a diseased gland in the groin of a negro, recovery following the excision of the gland; Prinz (4) reported a black yeast-like mold infection of the tongue; Alvarez (5) reported a red torula infection of the tongue. In 1927, Lloyd Jones (6) reported a torula infection involving the palate and nasopharynx, which apparently was arrested by careful cauterization and the therapeutic application of the x-ray, and the internal administration of potassium iodide. In the discussion of Jones' paper, Dr. Wm. D. Gill of Texas reported two cases, one of an ulcerative cutaneous lesion in the preauricular region on each side of the face which yielded to mild curettement, the x-ray locally and potassium iodide internally; the other infection involved the orbit and the sinuses of the head, and was accompanied by the presence of the torula histolytica in the blood stream, the condition apparently becoming arrested by the daily administration of 120 grains potassium iodide internally, 15 grains of sodium iodide intravenously and x-ray exposures to the orbit and sinuses. Including the two cases of Gill, twenty-four cases have been reported in the medical literature.

The group of blastomycetes known as *Fungi imperfecti* is composed of *torula, oidium, monilia* and *dematium*. Torula is considered to be a pseudo-yeast, occurring abundantly in nature, on trees, fruits, bees, wasp-nests and insects, and has been found in canned butter and in milk. Yeasts are found in the throats of many individuals and under proper conditions they become pathogenic, setting up inflammation of the mucous membranes of the throat and air passages, also producing more deep-seated infections of the tonsil or peritonsillar tissue with a predilection for the central nervous system and lungs.

REPORT OF A CASE

The condition was recognized by Dr. M. F. McCarthy of this city, who referred the patient to me for general treatment, after Dr. Geo. Rockwell had succeeded in isolating the organism from the lesion on the tongue following the methods employed by

Sheppe (7). Drs. McCarthy and Rockwell will make reports in special journals, listing special features and cultural characteristics of the organism isolated.

History—A man, aged 28, a laborer in a steel plant, suffered an injury to the anterior part of the tongue June 20, 1926, when a piece of hot steel flew into his mouth. He was given immediate attention by a physician, but this condition grew worse, the tongue gradually assuming the state as pictured in Fig. 1. He was an American by birth, of good family history, and had previously always been in good health except for an attack of acute appendicitis for which he was operated upon ten years ago, and for an attack of double pneumonia twelve years ago from which he completely recovered. He was married and had a healthy child. He had been examined for life insurance and accepted in April, 1926. The heart and lungs were found to be normal. Previous to the onset of the present illness, he weighed 206 pounds.

Examination—When I first saw him on January 11, 1927, he was considerably emaciated, weighing 146 pounds, felt weak and complained of pain in the tongue which caused inability to eat and sleep properly. He could speak with difficulty owing to the contraction of the anterior part of the tongue. His temperature was 100, and the pulse rapid. His appetite was good and he could only partake of liquid food. He was constipated as a rule. He complained of no headache or pains about the body except for a pain in the chest which he ascribed to pleurisy a week ago. He had no precordial distress; was dyspneic on exertion. There were no genito-urinary disturbances.

The pupils were equal and reacted normally. There were no symptoms to indicate involvement of the special cranial nerves. The lips were normal. The upper teeth were missing, those remaining in the lower jaw were in poor condition and there was evidence of pyorrhea. The anterior part of the tongue was smaller than normal and partly bound to the floor of the mouth. The anterior third of the tongue was distinctly ulcerated, would bleed easily and was the seat of numerous small whitish masses composed of mucoid-like material. The remainder of the tongue was coated white, with a small elevated patch on the right side, somewhat discolored. The tonsils were not enlarged or infected. There was no enlargement of the thyroid, submaxillary or cervical glands.

The chest was well developed, expansion being good. The left border of the heart was within the left mammary line, there was no dilatation of the aorta, no murmurs were heard, the action was rapid but regular. The examination of the lungs showed no alteration to percussion on either side; wheezing râles were heard throughout both lungs, both anteriorly and posteriorly, but no other râles were heard. Inspiration was somewhat roughened and expiration apparently somewhat prolonged throughout. Over the right base roughened grating sounds were heard, which were thought to be due to dry pleurisy. A fluoroscopic examination of the chest made at this time showed the heart to be normal in size and position; there was diffuse en-

largement of the glands at the hilum most marked on the left side; the parenchyma of the lung was mottled throughout; the costo-phrenic angles were clear; the diaphragmatic movements free and equal.

The examination of the abdomen showed no enlargement of the liver or spleen; at a later date the spleen was laris, infected teeth, diffuse pulmonary infiltration due to tuberculosis or torula infection, dry pleurisy of the right lower lobe.

Special Examinations

Urinalysis: Amber colored, clear, acid, specific gravity 1.010, sugar negative, albumin negative, diacetic acid

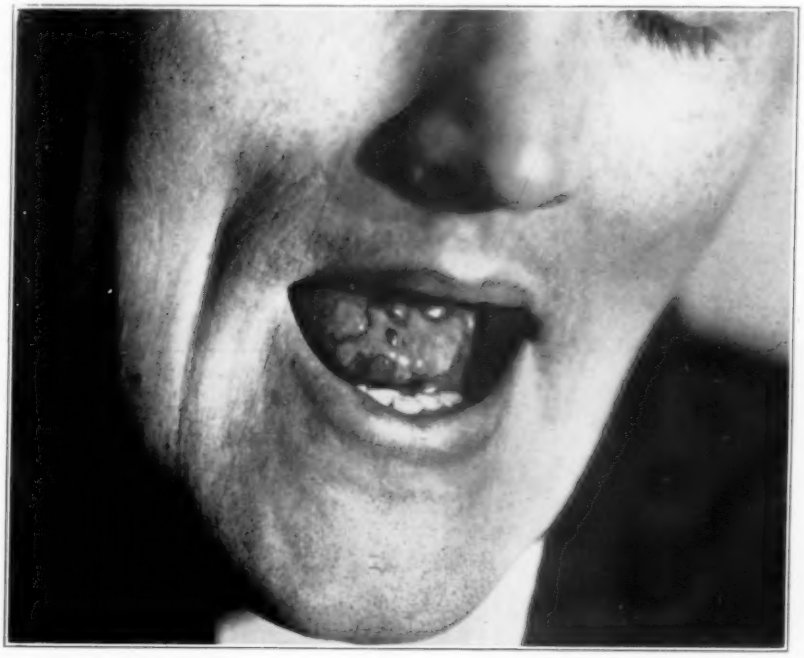

FIG. 1.—Showing ulcerated tongue from which the Torula histolytica was cultivated.

felt. There was no tenderness over the gall bladder or appendix areas; there was no palpable enlargement of the glands. The extremities were normal; reflexes active. The rectal examination showed no enlargement of the prostate or seminal vesicles; no hemorrhoids were found.

As a result of the examination the following diagnoses were made: ulceration of the tongue, pyorrhoea alveo-

negative, indican negative. Microscopic: no casts, no red cells, no pus cells.

Blood count: fresh specimen showed rouleaux formation, no change in the outline of the red cells. Hemoglobin 80 per cent. Red cells, 4,900,000. Color index 0.81. White cells 3,850. Differential: 54.5 per cent polynuclears, 30.5 per cent small and 5 per cent large lymphocytes, 1 per cent

mast, 3.5 per cent eosinophiles, and 5.5 per cent transitionals.

Blood culture: negative.

Blood Wassermann: negative reaction using cholesterinized and alcoholic antigens with the method of ice box fixation.

Kahn precipitin test for lues: negative reaction.

Tubercumet test of the blood serum: positive.

Intradermic tuberculin test: negative.

Dr. Rockwell had isolated the torula histolytica organism in October, 1926, using 0.3% malt extract, dextrose, and maltose with lactic acid media; also staphylococci and streptococci with plain media. A small piece was excised from the margin of the ulcer and examined microscopically, but no evidence of malignancy was found.

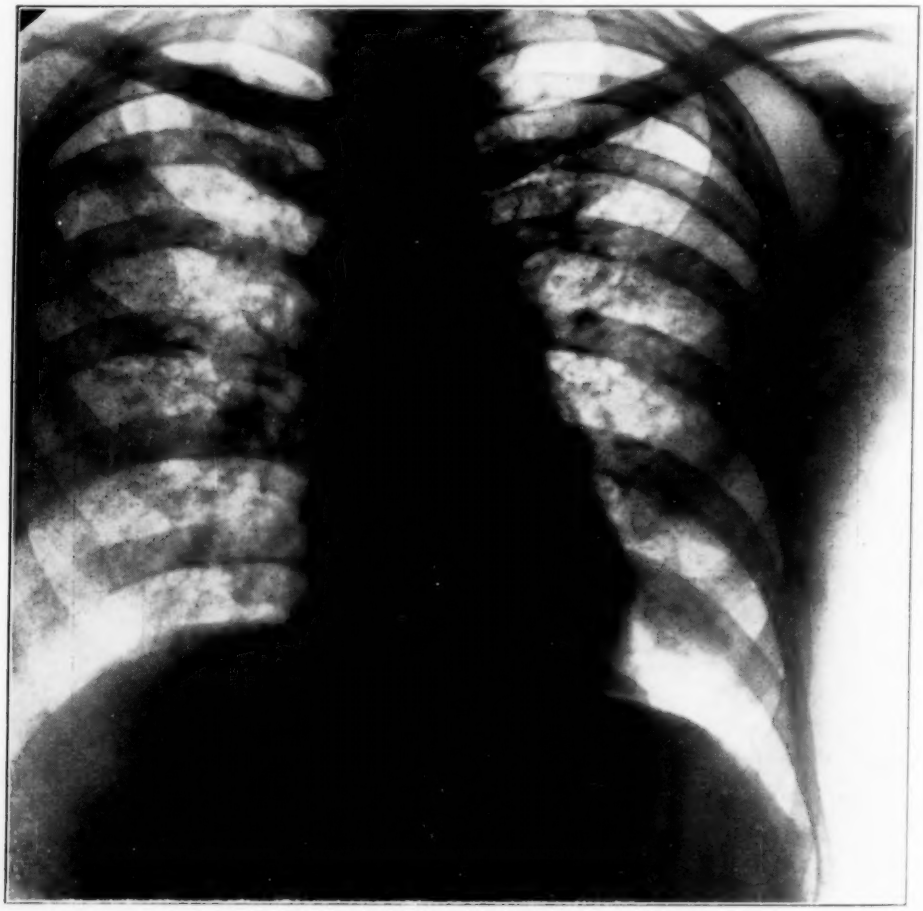

FIG. 2.—Stereoscopic picture showing diffuse bilateral infiltration, not typical of tuberculosis of the lung.

X-ray examination of the chest by Dr. Chas. Goosmann in January, 1927: stereoscopic pictures showed diffuse bilateral infiltration, not the typical picture of tuberculosis of the lung.

X-ray examination of the teeth by Dr. J. P. Becker, April, 1927: x-ray does not reveal evidence of tissue change about the apex; however, the right and left molars are converged and rather deep pocket formation about these teeth. The lower anterior teeth appear to be quite normal. The position of these teeth may be causing some irritation to his tongue.

Previous treatment and course: Local antiseptic measures including the use of mercurochrome, acriflavine, gentian violet, dioxygen, perborate of soda and arsenobenzol in glycerine; one fifteen-minute exposure to the x-ray, followed by an exposure of one hour to radium, which caused considerable local reaction. In addition the patient was given a course of subcutaneous inoculations, using a vaccine composed of staphylococci and streptococci isolated by culture from the tongue, but with no favorable results. A boiled aqueous extract of the torula culture produced a very marked local skin reaction when given subcutaneously. The course was progressively downward.

Subsequent treatment and course: Following the suggestions of Alvarez (5), the patient was given two grain doses of quinine sulfate three times a day, and two intravenous injections of 0.6 gram neo-salvarsan. Apparently the patient was much benefited; he was in a different frame of mind and more hopeful; the pain in the tongue disappeared and he could sleep better and eat solid food for the first time. The tongue began to improve; the temperature was 99.0. The pains in the chest disappeared, and the physical signs of the lungs had diminished, although the fluoroscopic examination showed the same infiltration. He now weighed 144 pounds. He was now given another intravenous injection of neo-salvarsan (0.6 gram) following which he grew worse. The use of quinine internally was continued and the internal administration of a saturated solution of sodium iodide, 15 drops three times a day, was begun. A week later general exposures with the ultra-violet mercury lamp were begun. The fourth and last intravenous injection of 0.6 gram ne-salvarsan was given four weeks after the third, followed by gastro-intestinal disturbances. A week later he again began to improve, both locally and generally. His weight remained about the same, 143 pounds, to April 5, 1927.

The period of improvement lasted about two weeks longer when there was a relapse, the condition of the tongue grew worse, the wheezing râles returned throughout the chest, the spleen grew larger and the temperature rose. An aqueous solution of acriflavine was used locally and 0.15 gram of the neutral acriflavine in 15 c.c. distilled water was given intravenously. There was no improvement. An injection of 0.3 gram neo-salvarsan was given intravenously on May 2, 1927. The patient grew steadily worse, the temperature rising to 101.4. He was now sent to a hospital and given a mild diathermy treatment locally to the tongue. The course was progressively downward, the patient

dying of inanition on May 17, 1927. We were very desirous of obtaining an autopsy, but this was denied. This was unfortunate in that a gross and microscopic examination of the lung and spleen in particular would have been instructive.

Discussion: A case of ulceration of the tongue is reported, in which the *Torula histolytica* was isolated from the lesion. The marked skin reaction following the subcutaneous injection of a boiled aqueous extract of a culture of the organism, indicates that a state of hypersensitiveness existed. Complications in the form of a mottled infiltration of the parenchyma of the lung and enlargement of the spleen, were present. The blood culture was negative. Unfortunately, permission was not granted to perform an autopsy to determine the character of the lesion in the lungs and spleen. At no time was it possible to obtain a specimen of sputum to examine for tubercle bacilli. The patient had never worked in an atmosphere containing dust, so that the changes in the lung cannot be attributed to siderosis. Temporary improvement followed the employment of neo-salvarsan intravenously. Local antiseptic measures in the form of the various dyes, arseno-benzol, and perborate of soda were of no avail. The exposure to the x-ray and radium was followed by such a reaction that the patient did not care to have these repeated. No improvement followed their use. Quinine sulfate and sodium iodide internally over a prolonged period seemed to be of no benefit. Death was due to inanition, the patient steadily losing in weight and finally refusing food.

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X-ray Treatment of Goiter

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THE indication for radiation treatment in goiter is hyperthyroidism, the contraindication is a normal or a minus metabolic rate. The exceptions to this rule are malignancy of the thyroid or inoperable intrathoracic thyroid. In the first instance, radiation may be used to supplement operative measures or as a primary treatment. In either case the prognosis is very grave. In intrathoracic goiter either with or without hyperthyroidism if operation is considered inadvisable, radiation should be instituted in an effort to reduce the size of the gland and relieve the pressure symptoms. In all other conditions of the thyroid gland radiation should not be used unless hyperthyroidism is present. If there is any doubt at all on this point the basal metabolism test should be the deciding factor. As a matter of fact, it is best to perform this test on all suspected thyroid cases. Personally, I do not treat thyroid cases unless they have a metabolic rate of plus 10 or more.

Hyperthyroids are commonly classified as (1) exophthalmic goiter and (2) toxic adenoma. For the purpose of discussing the treatment I will add to these (3) adolescent goiter and (4) hyperthyroidism without exophthalmos and with or without goiter. I am uncertain whether these two classes differ from exophthalmic goiter in any respect except that of severity. Surely one occasionally sees exophthalmic goiter in an adolescent but the great majority of hyperthyroids at this age, if untreated, will gradually recover without going into an exophthalmic stage or else will develop adenomata. In the absence of exophthalmos I question the propriety of calling such cases exophthalmic goiter unless it can be shown that the natural progress of the disease will eventually develop an exophthalmos. In the majority of cases of hyperthyroidism around puberty this does not happen, but it is still possible that these cases differ from exophthalmic goiter in degree only and not in kind. The same remark will apply to the classification which I have designated as hyperthyroidism without exophthalmos and with or without goiter. I referred to these cases in an article published in 1922 (1) and since then have seen many more. The widespread use of the basal metabolism test has brought to light an increasing number of these cases, although I would hesitate to accept a plus metabolism as an evidence of hyperthyroidism unless such a diagnosis could be substantiated clinically. These cases all have the exophthalmic goiter syndrome but none have exophthalmos and quite a few are without much enlargement of the thyroid.

I believe the hyperthyroidism is

probably the reaction to some toxemia, not necessarily always the same. Many of these cases have definitely pathological tonsils, some have infected teeth. In a number it is not possible to discover any such source of infection or irritation, but there may be conditions present which we do not recognize as etiologic factors. For instance, McCarrison believes that intestinal intoxication may be a cause of goiter. Whatever the cause these cases are definitely hyperthyroid and they do not always recover from the hyperthyroidism when the exciting cause is removed, although it goes without saying that this is the first thing that should be done provided the cause is known. Radiation treatment is particularly successful in this class of cases and surgery correspondingly unnecessary. I have no data at this time to show whether these cases would eventually develop exophthalmos or not. It may be that this is simply an early stage of exophthalmic goiter now recognized at such a stage because of the universal use of the metabolism test. However, reference to the histories of the cases reported in this paper show that 32 cases of undoubted exophthalmic goiter gave a total duration of 289 months or an average of 9 months before I saw them. Twenty-eight cases classed as hyperthyroidism without exophthalmos gave a total duration of 294 months or an average of 10½ months before applying for treatment. Such a comparison, of course, proves nothing, but one cannot help but wonder why this group of cases had not developed exophthalmos in such a length of time if they were ever going to. That all exophthalmic goiter cases are not due to one cause is proven by the fact that some come on suddenly after fright or shock and others develop gradually without any such etiologic factor. It is possible that the nature or severity of the etiologic factor may determine the development of exophthalmos, goiter, hyperthyroidism without goiter or adenomata. Perhaps the patient's inherent ability to combat the disease may bring about these variations.

It is generally accepted that the symptom-complex which is present in all the goiters which we are accustomed to speak of as hyperthyroids is due to an over secretion of the gland. However, this theory has been questioned lately by several authors, who suggest that the change in the secretion may be qualitative rather than quantitative. The Mayo Clinic advance the hypothesis that in toxic adenoma there is an increase in thyroid secretion in the blood, but that in exophthalmic goiter the character of the secretion is altered. Since the action of radiation on gland tissue is to produce an atrophy, if this theory is accepted, the rationale of radiation treatment might be seriously questioned. However, any one who cares to may watch a case of exophthalmic goiter being properly treated by X-ray and note the gradual subsidence of the so-called "hyperthyroid symptoms" and the corresponding fall in the metabolic rate. Whether this effect is due to an actual diminution in the quantity of thyroid secretion or to the destruction of gland cells which are throwing out a perverted secretion, the action on the patient is certainly beneficial.

The first step in the treatment of any disease is a correct diagnosis and too much emphasis cannot be laid upon the necessity of determining if a supposed thyroid case is suffering from hyperthroidism. The symptom-complex is well known and consists of nervousness, tachycardia, goiter and exophthalmos. To these major symptoms may be added weakness, loss of weight in spite of good appetite, sleeplessness, digestive symptoms, profuse sweating and a sense of profound anxiety which is practically always present. These characteristic symptoms added to an increased metabolic rate make the diagnosis. I do not think a diagnosis of hyperthyroidism should be made on an increased metabolic rate alone, if these symptoms are absent, and would advise against X-ray treatment under such circumstances.

We will now consider the types of goiter enumerated in a little more detail.

Adolescent goiter—The physiological enlargement of the thyroid gland at puberty should never be mistaken for a pathological condition. It is practically always present in young girls at this age and calls for no treatment whatever. However, a small percentage of these children develop hyperthyroidism and occasionally true exophthalmic goiter. If the hyperthyroid condition is recognized and a small amount of X-ray treatment given the prompt recovery of the patient is extremely satisfying. I have had no experience in the administration of iodine to prevent the occurrence of goiter in adolescents, but I have seen quite a number of cases in the last three or four years in which iodine was given to these little patients, who undoubtedly had nothing more than the normal physiological enlargement of that period, and a serious hyperthyroidism was brought on as a result of such treatment. As a matter of fact, this statement might be dilated on to include many cases of goiter in adults as well. There appears to be a widespread belief among the medical profession and the laity at large that iodine is a newly discovered and very effective drug in the treatment of goiter. Such is far from being the fact, as it is one of the oldest remedies used in this disease and its range of usefulness is quite limited.

The remarkable effects from the use of this drug at the Mayo Clinic to prevent post-operative complications in exophthalmic goiter has apparently led many members of the profession to believe that this is the drug of all others to use in the treatment of goiter, although Plummer who originated the treatment and many others who practice it enthusiastically are emphatic in saying that it is a method of preparing the patient for operation and not a method of treatment for the disease. In the last two years I have seen a great number of patients whose goiters were undoubtedly caused by iodine or made much worse by its administration. Some of these patients took iodine on their own initiative and others were given the drug by their physicians. Lahey, in discussing a paper by J. De J. Pemberton on this subject, expresses my own views exactly. He says: "It is the duty of all who deal with patients with disease of the thyroid firmly to impress on the

public that iodine is not a method of cure in exophthalmic goiter but a method of preparing patients for operation." While on the subject of iodine a word might be said about its use as a preliminary to X-ray treatment. There seems to be an impression that its effect would be the same for this purpose as it would if used as a preliminary to operation. I have used it in a few cases for this purpose but without effect on the course of the treatment so far as I could see. The pathological examination of thyroids removed after preliminary iodine treatment shows a marked increase in colloid material and a decrease in the size of the secreting cells. This would, in all probability, be an advantage to the surgeon but hardly to the radiologist as colloid is unaffected by radiation. However, the number of cases I have treated in this manner is not sufficient to justify any positive conclusions.

Hyperthyroidism without exophthalmos and with or without goiter—These cases have all the symptoms of exophthalmic goiter including a high metabolic rate but no exophthalmos and often very little or perhaps no goiter. Of the 36 cases reported in this paper 5 had no recognizable enlargement of the thyroid, 29 had a slight enlargement and 2 had well marked enlargement. They are commonly regarded as atypical cases of exophthalmic goiter, but I wish to question the propriety of such classification, especially as they are almost as numerous as the cases with exophthalmos. I feel certain that the thyroid gland is secondarily involved from diseased tonsils in a fair proportion of these cases in much the same manner as the cervical lymphatics are. Such cases will often not improve from radiation of the thyroid alone but respond immediately if the tonsils are treated also. I suspect that abscessed teeth may be an etiologic factor and possibly focal infections in other parts of the body may also cause a reaction in the thyroid. A large percentage of these cases do well under X-ray treatment and surgery seems unnecessary except as a remedial agent for primary foci of infection. The enlargement of the gland in these cases is similar in character to exophthalmic goiter and is not adenomatous.

Exophthalmic goiter—There appear to be three definite varieties of this type of goiter. (1) Those having a sex basis and occurring near puberty, pregnancies or the menopause. (2) Those with neurotic basis and caused by fright, shock, worry and so-forth. (3) Those in which the cause is undetermined. Although occurring from such different causes the disease is the same once it is established. It differs from the previous classification in that exophthalmos is a constant factor and the disease runs a typical course with remissions and exacerbations extending over a long period. It is differentiated from toxic adenoma by the character of the enlargement of the thyroid. In exophthalmic goiter there is a general enlargement either of one or both lobes, the gland is rather soft and may pulsate. In old cases, after repeated cycles of remission and exacerbation, adenomata are apt to be

present also. In adenoma there are one or more hard distinct nodules with well defined outlines.

In exophthalmic goiter, X-ray treatment is usually satisfactory, provided the case is seen before the first remission has occurred. After the case has gone through several remissions and exacerbations, the result is not apt to be so satisfactory. In 1917, I called attention to the fact that the longer a goiter had been present, the less likelihood there was of effecting a cure by radiation. My experience since that time confirms me in this opinion.

Severity of symptoms is not a contra-indication to radiation unless the patient is in a so-called "thyroid crisis," when she should, of course, be put to bed and given appropriate emergency treatment. Some of our most brilliant results have been in cases that had the most violent symptoms.

Toxic Adenoma—These cases are recognized by the presence of adenomata in the gland, combined with the symptoms of a thyrotoxicosis and an elevated basal metabolic rate. They are the least satisfactory of all hyperthyroids to treat by radiation in my experience. If operation is considered inadvisable, or refused, radiation should be tried, of course. However, I believe operation should have first choice, especially when one considers the possibility of malignant degeneration at a later date.

Technic of Radiation—This should be thought of primarily as a medical treatment and the same routine should be carried out as if the patient were going to be treated by medicine alone. That is to say, she should pay the same attention to hygiene, rest, diet, etc. I tell these patients that there are four things that are absolutely essential to their recovery: 1. Rest. 2. Fresh air. 3. Good food. 4. Sleep. Above all, the patient must be relieved from worry and excitement. In the matter of food, red meat is not allowed; tea, coffe, alcohol and tobacco are also prohibited. Drugs are only given for special indications and not to combat the disease. It usually requires about six months to bring the case to a point where treatment can be stopped and the patient should be told this at the outset.

As regards dosage, I am in the habit of applying one-half an erythema dose of rays of a quality corresponding to a 9″ parallel gap-filtered through 6 millimeters of aluminum, to one lobe of the thyroid each week. This gives a treatment over each area every two weeks. After two treatments are given to each area, the patient is given a rest from treatment for two weeks. This process is repeated as long as the treatment is continued. This is rather mild X-ray treatment and is not apt to produce skin changes or other untoward effects. Unless the patient comes from a distance and one wishes to save them the added exertion of frequent visits, it is wise to treat only one area each week rather than both areas every two or three weeks. By this method one has better supervision over the patient. This is quite important in patients as mentally upset as hyperthyroids whose daily habits and progress require the constant attention and supervision of the doctor.

Where radium is used, I apply half an erythema dose of gamma radiation

and repeat in six or eight weeks if necessary. I prefer X-ray to radium because I believe the regression is brought about more gradually and wtih less danger of producing ill effects by too rapid destruction of thyroid tissue. However, the cases in which I have used radium have all done very nicely.

If the case is no better after four months of treatment, radiation may as well be discontinued and some other form of treatment instituted. The average case usually shows a lowering of the pulse rate and a decrease in the nervousness after about a month of treatment, the improvement continuing gradually from that time. It is wise to follow the effect of treatment with the metabolism test and stop when the rate reaches plus 10 or thereabouts. There will be a continuation in the fall of the metabolic rate for some time after the treatment is stopped. The gradual lowering of the pulse rate may be utilized for the same purpose, the point being that treatment should be stopped before the thyroid reaches normal, or the cumulative effect of the radiation which continues for some time may induce a condition of hypothyroidism or myxoedema.

	Cured	Improved	Unimproved	Quit	Operated	Died	Still under treatment	Total	Incidence of diseased tonsil
Adolescent goiter	9			2	1			12	3
Hyperthyroidism without goiter and without exophthalmos	3	2						5	2
Hyperthyroidism with goiter and without exophthalmos	21	3		4			3	31	12
Exophthalmic goiter	21	4	2	9	4			45	18
Toxic adenoma	1	1		2	1			5	1
Total	55	10	2	17	6		8	98	36

Aneurysm of the Renal Artery

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THE chief interest of this case report is the interpretation of the curious roentgenogram, which readily permits the diagnosis to be made if the condition is thought of. A point of further interest concerns the etiology of aneurysm of the renal artery, as well as of arteries to the other abdominal viscera: syphilis does not seem to play the same important part as in aneurysm in general.

Case Report: L. T. A white woman, aged 59, was admitted to the Jefferson Hospital on the service of Dr. S. Solis-Cohen, November 21, 1925. She complained of cough, shortness of breath and swelling of the feet. Her family history was unimportant. Her general health had been good until the onset of the present illness. This began about two years before admission with dyspnea on exertion. Later she began to cough and prior to her admission to the hospital her ankles became much swollen. In the last two months her appetite had been very poor; the sight of food nauseated her and partaking of it made her vomit.

Physical examination, on admission, showed a thin woman, sitting up in bed, very much distressed. The pulse was 100 and irregular; radial and temporal vessels were somewhat thickened; the heart was enlarged to the left as far as the anterior axillary line and there was a slight systolic murmur at the apex but no thrill. There were a few râles at the bases of the lungs, and the liver was greatly enlarged, extending almost to the iliac crest. The ankles and legs were markedly swollen. We felt that this was an ordinary instance of congestive heart failure, depending upon advanced myocardial degeneration. After several days in bed she was greatly improved. The electrocardiogram indicated branch bundle block and myocardial degeneration. The urine contained a trace of albumin, hyaline and granular casts, but no blood or pus cells. The blood urea was 13; the blood Wassermann negative. In spite of improvement the liver remained as large as before and now we made out beneath and below the liver a firm and tender mass. This was thought to be kidney or gall-bladder. However, gastric distress continuing, a roentgen-ray study was ordered, with the interesting result shown in the accompanying plate; a calcareous mass or masses clearly outlined at about the level of the 2nd lumbar vertebra. Suggestions regarding this curious finding were,—calcareous gall-bladder; cyst with calcareous deposits in the wall; or enterolith.

The patient failed to maintain her early improvement and gradually became weaker. Her cough became more troublesome and in the last few days of life she spat up considerable blood.

She died on December 17, 1925.

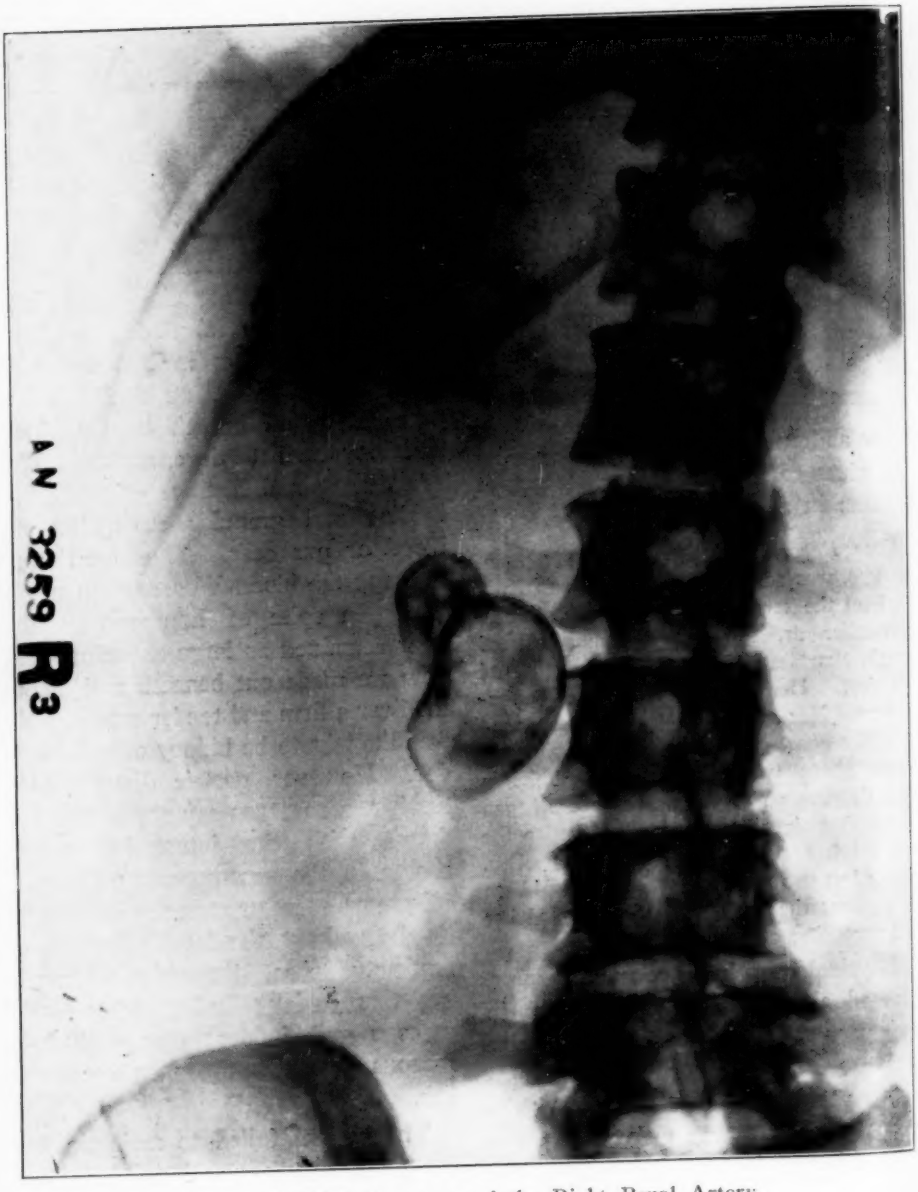

FIG. 1. Calcified Aneurysm of the Right Renal Artery.

Post-mortem examination (Dr. C. J. Bucher) showed an hypertrophied heart with myocardial fibrosis, large infarcts at the base of the right lung, a large "nutmeg" liver and a calcified aneurysm of the right renal artery, pressing upon the duodenum and pushing the kidney down. It was the latter organ that we felt as the firm tender mass below the liver. The right kidney contained a number of large anemic infarcts. The aorta was the seat of advanced atherosclerotic change but there was no evidence of syphilis.

COMMENT

Aneurysm of the renal artery is a rare form, constituting about 1.2% of all aneurysms. A total of 40 cases had been reported up to December, 1926 (1). The etiology is especially interesting. Among 31 cases collected by Conroy (2), 17 were traumatic and 15 non-traumatic. Of the latter 6 were associated with arteriosclerosis and 9 followed severe infections. In this connection attention may be called to the similarity of etiologic factors in aneurysms of arteries to other abdominal viscera. Among 65 cases of aneurysm of the hepatic artery collected by Friedenwald (3) in 1923, arteriosclerosis was present in 12, acute infections such as typhoid, pneumonia and osteomyelitis apparently were responsible in 18, trauma in 8 and syphilis in 7. The same seems to be true of splenic artery aneurysms. Trauma, acute infections and arteriosclerosis play a greater role than syphilis in the causation.

Renal artery aneurysm of the type reported above is usually a pathological curiosity. It is of arteriosclerotic origin, frequently calcified, does not erode and rarely ruptures. It is usually unattended by symptoms except that it may occasionally be responsible for infarcts of the kidney. As in the present instance it may be discovered incidentally by a roentgen-ray examination. It is questionable whether under such circumstances the patient should be subjected to operation. If the condition of the patient is good operation might be attempted, but if not the aneurysm in itself is no more dangerous than in other portions of the diseased vascular system.

A case similar to this one was reported recently by Sűnderlund (4). A woman of 61, had symptoms of colon pyelitis for 2 years. The condition had grown worse with recurring attacks of stitch-like pain in the region of the left kidney. Cystoscopic examination showed slight cystitis. Colon bacilli and pus were found in the urine. The roentgen-ray showed a ring-shaped stone shadow, about half the size of a walnut, in the hilus region of the left kidney and calcific deposit in the wall of the aorta. It was proved by pyelography that the shadow was outside of the renal pelvis but very close to it. The correct diagnosis of calcified aneurysm of the renal artery was made. (A previous case had been studied in the same hospital several years before.) The diagnosis was supported by attacks of pain in the left kidney region which were interpreted as due to infarcts. Nephrectomy was performed. A calcified true aneurysm, 15x12 mms. in size and containing a thin layer of thrombotic material, was found on the upper main branch of the renal artery together

with several small infarcts of the kidney. The patient was discharged in good condition.

This seems to be the first case in which the diagnosis was made prior to operation or section. The diagnosis was made possible by a consideration of the following points:—

1. The likeness of the X-ray to the previous case studied in the same hospital.

2. The marked arteriosclerosis of the patient with calcific deposits in the abdominal aorta.

3. Stitch-like pain interpreted as due to infarcts of the kidney.

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The Value of Scholarship*

By Chas. Gordon Heyd, B.A., M.D., F.A.C.S *Professor of Surgery, New York Post-Graduate Medical School and Hospital*

SIXTEEN years ago I graduated in medicine and am returning tonight to acknowledge my great obligation to the Medical Department of the University of Buffalo.

You may remember that when Dante and his companion had climbed, with considerable toil, up the Mountain of Purgatory they approached a wide and pleasant looking terrace encircling the hill and being tired they sat down to rest and Dante, looking back upon the steep ascent to the terrace upon which they rested, turned to his conductor and said, "All men like to look backward."

If one could dispassionately examine the period of preparation and the subsequent augmentation of his medical knowledge he would at least approach the subject in a frankly critical frame of mind for the sole purpose of evaluating what he considered most essential in the progressive development of his professional capacity. The physicists will tell you that action and reaction are always equal. In my own particular case the benefits that I have derived from studious application to the tenets and principles of my profession have been more than the effort, concentration and intensity of study would seem to warrant.

In addition, my remarks may be taken as the confession of a comparatively young man engaged in the practice of surgery, one who has to some extent been disillusioned, but not unhappily, and who has played the game as he has found it and whose experience may not be without some value to those who are junior to him in experience.

It has been recorded that three men set forth in search of Truth. The first said, "I will go to seek it in the wisdom of others. There, if anywhere, it is to be found." So he ensconced himself in the alcoves of a great library and began to pore over the tomes of all the sages in all centuries. Years and years he delved thus. One day as an old man he abruptly closed the volumes spread about him. "They all contradict each other," said he. "There is no such thing as Truth." The second man said, "It is not in books but in the lives of my fellow men that I shall find Truth." So he went forth for the quest, through all the marts and byways of mankind. In a few years he returned wearing the cynic's sneer. The third man hesitated. Said he, "Before I go to seek Truth, I fancy it will be well that I should try to practice it myself." And

*Delivered before the Epsilon Chapter of Alpha Omega Alpha Fraternity, Buffalo, New York.

in that endeavor he became so absorbed that he postponed his great quest, year after year, all his life long. As he lay upon his deathbed, he sighed deeply. "I vowed to seek for Truth," he murmured, "and I have broken my vow." And then, even as he raised his eyes, there stood before him a shining apparition of great beauty. "I am Truth," said the figure and smiled down upon him. And, as the man gazed in silent amazement, the voice continued, "Truly, you had no need to look for me, for you found me and I have been at your side since that day long ago when you chose to do rather than to seek."* Your entire professional life will be one of action and it is essential that you live and act the truth.

The essence of science is change and its function progressive improvement. As we continue to add to our professional activities from day to day we undergo a successional development in our medical knowledge and skill. Each new endeavor in scholarship is a milestone marking the way and if we could survey these milestones with a sufficiently distant perspective they would seemingly make a continuous straight line of progress. We know this is not strictly true, for life is ever a circle in which the present hearkens back to the past. Michael Angelo in Longfellow's poem expressed the thought so eloquently and so beautifully:

What other things I hitherto have done
Have fallen from me, are no longer mine;
I have passed on beyond them, and have left them
As milestones on the way. What lies before me,
That is still mine, and while it is unfinished
No one shall draw me from it, or persuade me
By promises of ease, or wealth, or honor,
Till I behold the finished dome uprise
Complete, as now I see it in my thought.

*Editorial, Collier's Weekly.

The four years of study in the Medical School which culminated with the degree of M.D. were but a stage in the preparation. You and I were called upon to digest and assimilate a great many unrelated facts. These were combined with a variety of theories and it was by the mental capacity and practical turn of our minds that this information was made useful in its most efficient form. The mere acquisition of the knowledge of the undergraduate course would have possessed relatively little value if it were not closely identified with the recognition of the clinical manifestations of disease. To have passed your medical examinations as an honor man or to have obtained a cum laude degree would have been relatively unimportant if the scholarship which it represented had not been continuous. After all, the science of medicine changes and moves on to new things. Our conceptions of histo-pathology are changing from year to year. Our therapeusis is being rewritten. The door of surgery is ever opening wider and wider. It is essential that all of us undergo a continuous or periodic re-education in medical knowledge, and from time to time, by travel, by post-graduate work, by selective reading, evaluate our clinical information in terms of present day scientific knowledge.

A bright and capable young man

could undergo the most extensive preparation in surgical laboratories and hospitals and fail to be even a modest success as a scientific surgeon. A man may by technical imitation become an excellent operator or he may make rounds through the hospital wards and in a mechanical way apply the principles of medical practice. This implies that the practice of medicine or the specialties may be purely mechanical as distinguished from the art of medical practice. Some men are endowed by birth with qualities that eventually make them successful doctors. Many will acquire the clinical personality that will make successful practitioners. It will be given to a few to have, either by natural inheritance or by the peculiar coaptation of personal qualities, that particular something that represents the art of practice. The art will connote that the individual is a teacher and a trainer of young men. The actual amount of labor that any one of us can accomplish as represented by the numerical notation of operations, sick people that we have attended, or medical contributions throughout an active lifetime is obviously small, but to the teacher in medicine his contribution is represented by the sum total of the labors of his students. To be a teacher demands a solid foundation of well qualified scholarship, character, good health and much study. If I should select an epigram as a descriptive caption for a teacher I should select the somewhat paradoxical phrase "the joy of labor." When the divine fiat was given to mankind, "by the sweat of thy brow thou shalt labor," there was expressed the greatest panacea for unhappiness. When man is truly happy he labors most diligently in the particular vocation to which he has applied himself.

I should like to believe that aside from the patients that have been benefited and lived longer in happiness and contentment by reason of my surgical ministrations, I was essentially a trainer of youth. I know that here and there throughout this country are former students of mine who have received a little more knowledge, whose paths have been made easier, the inspiration for service more adequately instilled, the necessary self-denial and fortitude encouraged and that from this personal contact there flows that contentment which comes from being a teacher. A good teacher is scholarship successfully applied. As the mechanism, mental and physical, that most contributed to scholarship I place first the value of being born with a good heredity and a strong vibrant body. While an infirm or weakened constitution may carry a superlatively fine brain the demands made upon you will almost without exception require a splendid physical equipment. I would place second the master word "Work." Work, not for what the labor actually and by itself produced but what flowed from its application. Luck and chance may contribute no small part in placing you at the most opportune time in a position of great personal and professional advantage, but the ability to seize, hold and accept these opportunities will be the result of a preparation which has been intensive, prolonged and ofttimes painful. The formula may be adduced that work creates the ability to visualize and accept opportunity.

The casual practice of medicine is not enough to enable the young practitioner to carry himself through life successfully. He must learn how to use his education, to apply what he has learned to the everyday experience of his practice and to translate his medical information into terms of diagnosis, therapeusis and prognosis. In the application of this information in clinical medicine, irrespective of his specialty, he will suffer many disappointments because diagnosis must ever remain somewhat inexact by reason of the fact that we are interpreting along with our clinical facts and symptoms a diseased psychology. In the orderly rearrangement of his knowledge with such additions as science contributes to medicine from time to time he will derive his pleasure and his maintenance. He will be fulfilling in a large way the dictum of Shaw that an individual is entitled to as much happiness as he creates and no other activity will yield him such a large measure of return as adequate scholarship in medicine.

To put your name to an immortal poem or manuscript is genius; to be in a political position to write a pardon is power; to have financial resources sufficient to make good a check is capital; to have an understanding of the tools and equipment of your profession is happiness.

We live happily or otherwise in an age and in a country where the materialistic phase of success receives adulation and incites more imitation than any other. The more delicate, refined or subtle tests of success, success in a spiritual sense, success in the adjustment of oneself to the world about him, which appraises service rather than gain, success which recognizes self-denial is often placed secondary to great wealth and power. In the acquisition of money I do not believe one can model a really successful career as a doctor, and I think if one will calmly survey his own activities he will be apt to believe that success lies in the development of character and self-control through personal experience.

There is no nobler calling in the annals of human affairs than that of a true practitioner of medicine. He must play the game at all times, sometimes under the most disadvantageous of physical circumstances, and the worldly remuneration he receives is, as a rule, inadequate to his self-devotion and sacrifice, but he has the satisfaction that comes from the thought that he has devoted himself to a service that, irrespective of the financial and social remuneration he will derive, gives a larger measure of happiness than can come from any of the other more or less arbitrarily applied professions.

The mere acquisition of money to the majority of people is probably conceived as the most useful occupation for the employment of one's talents. Yet to have obtained an excess of money and to have contributed nothing in the upbuilding of your students or of your profession would be indeed but a shallow measure of success. It would be absurd to suppose that money has no place in the life of a successful doctor. While art may rear itself from the sordid, meagre environment of poverty, the successful practice of medicine demands that the phy-

sician shall be relieved from the harassment and pressing demands of financial worry.

It is doubtful if any of us, when we began our medical course, had a well-defined objective. We were probably wise enough for our day to assume that a moderate amount of labor would give us adequate financial stability and the various callings of our profession provide sufficient labor to maintain a fair degree of contentment. As we grow older and see the number of men who have, according to our own opinion, failed, we cannot help but recall Goethe's hard saying, "Death is nature's expert advice to get plenty of life."

It is wise that all of us should determine for ourselves the final objective of our endeavors. We must of necessity feel that the practice of medicine in some one of its specialties is, after all, our vocation. We should zealously strive to perfect ourselves in the fundamentals of our science. It is well not to be too fixed in one's idea as to the exact established boundaries of medicine. Medicine embraces so many sciences and has so many angles, is capable of being applied in so many ways, that neither opinionativeness nor obstinacy of practice should make us forget that a large number of men go through life unchanged by the orderly progression and extension of the boundaries of medicine. How often have all of us heard:

"The sad rhyme of the men who proudly clung to
Their first fault and withered in their pride."

It is essential for a person to cultivate a hobby, and I think for a medical man the cultivation of some type of literary pursuit, aside from one's reading in medicine, is the most profitable hobby or avocation that one can have. It requires but a little time, conscientiously given day after day, to acquaint oneself with the best and most splendid things in this world of ours, and no profession should be so crowded or the exigencies of practice so exacting that it does not permit time for the dreams that precede action.

"Books are the tools of the mind. Their function is to increase the treasures of wisdom and knowledge. In a very high sense they are the true labor-saving devices. What the loom does for the fingers, what the engine does for the feet, what the telescope does for the eye, that and more, books do for reason and memory. They hasten man's intellectual steps, they push back the intellectual horizon, they increase the range of his vision, they sow intellectual harvests otherwise impossible, and they reap treasures quite beyond the reach of the unaided reason."*

*Newell Dwight Hillis, in the Watchman-Examiner.

Editorial

Tobacco and Physical Efficiency

THE Committee to Study the Tobacco Problem published in 1923 Professor O'Shea's book on "Tobacco and Mental Efficiency," and in 1924 Professor Clark L. Hull's monograph on "The Influence of Tobacco Smoking on Mental and Motor Efficiency". In addition to these publications a number of laboratory studies have been published independently. The author of the present work just brought out under the auspices of the Committee is Dr. Pierre Schrumpf-Pierron, Professor of Clinical Medicine, University of Cairo, Egypt. There is a preface by Henri Vaquez, the noted Professor of Medicine, University of Paris. The members of the Committee, fifty-two in number, are well-known physiologists, internists, psychologists, pharmacologists, cardiologists, hygienists, geneticists, physical educators, economists, medical directors of life insurance companies and others. Alexander Lambert is President of the Committee. The publications of this body are presented in an unbiased manner, and the variations of opinions offered are those of the author or investigator, the committee itself expressing no opinion of its own as to the effects of tobacco on the animal organism. It has not attempted to give the results of original scientific investigations of its own, but presents a succint survey of the literature, accompanied by an extensive annotated bibliography. There is a very great mass of clinical material bearing upon the physical affects of tobacco, but it is widely scattered and much of it inaccessible. It has been very difficult for any one interested in the medical study of tobacco to get at this material, and there exists no medical compilation or analysis of clinical observations or studies along this line. Most of the lay writings about tobacco are prejudiced one way or the other, and very few physicians when pressed for definite statements as to the effects of tobacco upon the body are able to make any satisfactory statement. The problem is very much more difficult than in the case of alcohol, and various medical committees, notably that of Great Britain, have collected and analyzed the facts as to the effects of alcohol, and have made concise statements regarding these, accessible to every one. The pathological effects of tobacco, on the other hand, are not so apparent, and there is much greater opportunity for differences of opinion. There is much active propaganda for and against tobacco; both sides need the facts, and the subject needs thorough investigation and discussion. This book presents very complete data of all that is known about the action of tobacco by scientific investigation and observation up to the present time. The various chapters

cite the bibliographic references, state the nature of the investigation and its results, and give a brief summary of the total evidence offered. It is an unbiased and truthful discussion of one of the important problems of the day. The collective evidence as to the effects of tobacco as summarized in this volume are as follows. The chief poison in tobacco plant leaves is nicotin. In considering the toxicity of tobacco, however, the combined effect of nicotin and other components formed in the process of fermentation or due to combustion must be taken into account. Among these the first in importance are pyridin, ammonia and collidin. Prussic acid, carbon monoxide, pycolin, the phenols, etc., have but a secondary and perhaps negligible effect. Acute tobacco poisoning is due mainly to the effects of nicotin, although pyridin and other toxic constituents may have some influence. It is characterized by pallor, dizziness, faintness, sweating, palpitation, muscular weakness and collapse. Nicotin first stimulates and then powerfully depresses both the sympathetic and the parasympathetic ganglia of the autonomic nervous system; it also stimulates and then depresses the central nervous system, and the endings of the motor nerves in voluntary muscles. When injected into the circulation nicotin first causes a transient elevation of blood pressure and slowing of the pulse. These effects are due to stimulation of the vasomotor center and ganglia. With large doses the secondary effects on the circulation are a fall of blood pressure and marked quickening of the pulse, due to depression of the peripheral vasomotor and cardio-inhibitory mechanisms. Nicotin first stimulates and then depresses the respiratory center. Large doses produce nausea and vomiting, partly through peripheral action, but chiefly through stimulation of the medulla. Death has occurred from the use of 30 gms. of smoking tobacco and 12 gms. of snuff. According to Schmiedeberg the fatal dose of nicotin for a non-smoker is the quantity contained in two cigars of moderate size, provided they could be swallowed entirely. Tolerance is easily acquired and habitual users of tobacco may absorb comparatively enormous doses and may easily tolerate from 40-50 mgms. of nicotin per day. There is no complete and trustworthy work on the comparative toxicity of different kinds of tobacco. Measurements of the amount of nicotin retained in the smoker's mouth, swallowed or absorbed, show, according to Lehmann that from one gram of cigar, there is retained or absorbed from 0.3 to 0.8 mgm. of nicotin; from one gram of cigarette 0.4 to 0.5 mgm. will be retained. As to its action upon the central nervous system there is reason to believe that chronic poisoning may give rise to motor and sometimes sensory aplasia, hemiparesis, and hemianesthesia; occular disturbances, such as retrobular neuritis, hemianopsia, floating specks, inequality of the pupils, sluggishness of the external muscles of the eye; disturbances of the cochleovestibulary nerve and the auditory and olfactory nerves, and neuritis of the cerebral nerves (facial, trigeminal and hypo-glossal). Neuralagias, both diffuse and isolated (brachial, occipital, sciatic,) true polyneuritis, and finally symptoms of motor irritation (trembl-

ings and twitchings) are also very frequent in tobacco users. Tobacco may also cause headache, migraine, vertigo, insomnia, mental depression and amnesia. The abuse of tobacco is the cause of many cardio-vascular neuroses and disturbances of heart rhythm. Secondarily, it may bring on hypertrophy of the heart. There is a conflict of opinion as to its possible role in the causation of actual lesions of the heart or blood vessels. A verdict of "not proved" on this question must be given, but there seems to be unanimity of opinion as to the adverse effects of tobacco in cases with latent or active changes in the cardiovascular system. Upon the respiratory system the effect of nicotin in small doses is to excite the respiratory center, moderate doses first excite and then paralyze, while large doses immediately paralyze it, death taking place from suffocation. Smoker's chronic laryngitis and smoker's asthma are well known clinically. The symptoms of the latter, evidently due to incipient paralysis of the center, disappear when the patient ceases smoking. In the digestive tract chronic pharyngitis due to the irritation of the smoke is very common in smokers. Less important is the so-called smoker's dyspepsia. Hyperchlorhydria is common in heavy smokers, and achlorhydria in chewers. The digestive disturbances of smokers are of nervous origin due to irritation of the centers. No antiseptic action of tobacco smoke on the mouth cavity is produced; carious teeth appear to be more frequent in smokers than in non-smokers. It is curious that this report does not discuss the question of the relationship of smoking to lip cancer since this etiology is being stressed so heavily at this time in England. Buccal cancer is noted, and the conclusions drawn from the literature are to the effect that the role of tobacco in its production has been exaggerated. The action of tobacco upon the genitourinary system is practically unknown, and various statements as to the relation of smoking to impotence, miscarriage, metrorrhagia, etc., rest upon a doubtful foundation. On the other hand, the effects upon women workers in unhygienic tobacco factories appear to be more definite. Bottstein believes that certain forms of pruritus are associated with the abuse of tobacco. The role of tobacco as an important secondary factory in syphilis, alcoholism, lead poisoning, etc., is well known. The effect of alcohol upon the nervous system is greatly intensified by the use of tobacco; this is also true of the cardiovascular system. The chronic use and abuse of tobacco is frequently a complicating factor in neurosyphilitics. The psychological effects of nicotinism have been widely discussed. There appears to be a generally unfavorable reaction of smoking upon scholarship, and upon the general mental efficiency, the greatest losses being in the fields of imagery, perception and association. Nicotin does not stimulate the cerebral functions, the contrary is the case. Smokers who attribute to smoking an increase in their capacity for intellectual and physical work are subjects of auto-suggestion, or the increased cerebral circulation from increased heart frequency may seem to produce an increased mental activity. It is curious that heavy smokers do not like to smoke in the dark, and that few blind people smoke. During the

war it was often noted that heavy smokers on losing their sight as the result of a wound, ceased smoking after a few months. The purpose of this work of the Tobacco Committee has been to collect the recorded medical knowledge regarding the effect of tobacco upon the human organism. As the result of this attempt one is compelled to admit that from the scientific and clinical point of view the study of the action of tobacco, as is also that of alcohol, must be still regarded as incomplete. The problem is very much the same in regard to both alcohol and tobacco. A sound individual may bear what for him is moderate doses without injury, but these amounts may be harmful to the unsound or to other apparently sound individuals. The immoderate use of tobacco brings on a series of disturbances which are at first functional, then organic, and some of these are of serious importance. Since the War, and the excessive use of cigarettes following it, these clinical disturbances have become much more important and more frequent. The cigarette habit leads readily to abuse, and the cigarette smoker usually consumes more tobacco in smoking cigarettes than in smoking cigars. Further, because of the frequent habit of inhalation of cigarette smoke an opportunity is offered for the greater absorption of toxic substances. It is highly desirable that carefully controlled laboratory and clinical studies be carried out regarding the effects of the prolonged moderate use of tobacco, as well as its free use.

Abstracts

An Experimental Study of Diathermy. By Carl A. Binger, M.D., and Ronald V. Christie, M.B., Ch.B. (Journal of Exper. Med., October 1, 1927, pp. 571-600.)

In view of the increasing use of diathermy by practitioners as a therapeutic measure, and as we do not possess any very definite knowledge concerning its effects upon internal tissues and organs it is very essential that investigations along this line be conducted until we have as thorough knowledge as possible of the mode of action, effects and sequelae of this use of heat. Binger and Christie have attacked this problem, and have published so far three papers: I. The Measurement of Lung Temperature, II. The Conditions Necessary for the Production of Local Heat in the Lungs, III. The temperature of the Circulating Blood. They were led to a study of the bodily responses to high frequency currents as a preliminary to an investigation of the value of diathermy in pneumonia. The problem confronting them was to find out whether deep localized heat could actually be produced in the body. The heating effects of the diathermy current have for the most part been studied in non-living systems; a favorite experiment has been the coagulation of egg albumin, or the cooking of meat and potatoes. The living body is not a sausage, nor yet a tube filled with albumin water, but is a heterogeneous system composed of tissues with different specific conductivities and heat capacities. Thus far, it has not been definitely established either that deep local heat can be produced by the diathermy current, or indeed, that the current penetrates into the deeper tissues of the body. Evidence has been presented that the so-called "skin effect" is a factor in keeping the current near the surface of the body. Bethman and Crohn state that they found it extremely difficult in experiments on anesthetized dogs to raise the systemic temperature more than a few fractions of a degree. The fact of the passage of current through tissue can best be established by proof of heat development in the tissue. That this heat may be quickly dissipated, and that it may be conveyed to the tissue from adjoining structures, are two complicating factors which need to be carefully controlled. Working upon anesthetized dogs the authors found that evidence was obtained to show that in normal animals the rectal temperature can be elevated by the passage of high frequency currents. During life the intraabdominal and intrathoracic temperatures can be increased only slightly above the rectal temperature. The lung temperature in the anesthethized dog normally was 0.3—0.4 below the rectal temperature. During the passage of diathermy currents of strengths equivalent to those used in therapy this relationship is reversed—the lung temperature exceeding the rectal temperature by about the same value. Immediately after death, the temperature rises abruptly in the deep tissues between the electrodes. For the measurements of deep temperature special thermocouples were devised by the investigators, and their method of preparation and mode of use are described. In their investigations of the conditions necessary for the production of local heat in the lungs, it seemed apparent that blood cooling is a more effective mechanism in removing heat from the lung than air cooling. Indeed, it is probable that the condition most needed for the production of local heat in the lung, by the passage of the diathermy current, is a partially or completely blocked circulation in the pulmonary and bronchial vessels. Prevention of the access of air to one lung, while its circulation is intact, results in little, if any, change in the rate of heating of the lung by the diathermy current. Occlusion of a main branch of the pulmonary artery during the

flow of the current results in a sudden rise in temperature in the lung where the artery has been occluded, with subsequent heating, however, at the original rate. Under these circumstances death of the animal is accompanied by a precipitous rise in the temperature of both lungs. When the pulmonary veins as well as the artery to one lung are ligated the circulation through bronchial vessels is also stopped. Diathermy then results in a local rise in temperature in the lung equivalent to that seen in the other lung after death. These experiments convinced the workers that the normal lung can be heated only slightly above the systemic temperature by the application of high frequency currents to a dog's thorax. Interference with the circulation of blood, however, provides the conditions necessary for local deep heating. The implication seems obvious that the circulating blood carries away the heat produced in the lung. Such an interpretation is in harmony with the physiology of heat distribution and heat regulation. It is probable that together with its many other equilibrating functions the blood is a fairly ideal medium for distribution and maintenance of a uniform temperature. Further evidence of the effective cooling of the lung during diathermy by the blood circulating through it is presented in their third paper. It can be estimated roughly, assuming a minute volume of blood flow through the lungs of 2.50 liters, and a rise of 0.2°C in arterial blood temperature above venous, that approximately half of a large calorie of heat is being removed from the lungs per minute. This is evidently sufficient to prevent any marked degree of local heating. In conclusion, these investigators have devised a method of measuring intravascular temperatures in anesthelized dogs. The temperature in the abdominal aorta is uniform throughout, and varies only with the systemic temperature. The temperature in the inferior vena cava rises as the thermocouple approaches the heart, reaching its maximum at about the level of the hepatic veins. Between the hepatic veins and the right chambers of the heart there is no further elevation in venous temperature. The temperature of the right heart blood normally exceeds that of the left heart blood by 0.05—0.2°C. During the application of high frequency currents to the thorax, this relationship is reversed. This indicates that the lungs are being heated, but that the blood passing through the pulmonary vessels is removing the heat at approximately the rate of production.

The Treatment of Hemorrhage in Peptic Ulcer. By WALTER A. BASTEDO, M.D. (New York State Journal of Medicine, 1927).

Hemorrhage from a peptic ulcer may be: 1. a slight oozing which appears merely as occult blood in the stools; 2. a larger hemorrhage which shows grossly in the stool but it not large enough to produce obvious symptoms of hemorrhage; 3. profuse hemorrhage accompanied by weakness, fainting, air hunger and anemia; 4. continuous or frequently recurrent small hemorrhage that gradually brings on a marked secondary anemia. Whether the ulcer is gastric or duodenal does not alter the medical treatment for hemorrhage. The therapeutic desiderata are stoppage of the bleeding, overcoming the effects of loss of blood, and the fore-stalling and prevention of hemorrhage. His summary of treatment in severe hemorrhage: 1. Have the patient very quiet, lying down with head low, with plenty of fresh air and with body kept warm.

2. If there is severe exsanguination, bandage legs and arms, raise the foot off the bed, and bandage and put weights on the abdomen.

3. Avoid unnecessary manipulation of the abdomen.

4. Give a hypodermic of morphine sulphate 0.015 grm. (gr. ¼) with strychnine sulphate 0.002-0.003 gram. (gr. 1/30-1/20).

5. Administer hypodermically every six to 12 hours 20 c.c. of fresh rabbit or horse serum 2 c.c. of hemostatic serum or 10 c.c. of thromboplastin.

6. Prepare early for transfusion and as soon as there are indications for it transfuse.

7. If there is exsanguination to the danger point and transfusion cannot be done, give intravenously Locke's or Ringer's solution, normal saline or 20 per cent glucose, and

give normal saline by rectum or by hypodermoclysis.

8. If the stomach remains distended and the bleeding seems to persist, lavage with tepid water and follow this by passing in through the tube a solution of thermoplastin, kephalin or gelatin with epinephrin. In cases with portal congestion, or if the bleeding has apparently ceased, avoid lavage.

When these measures are unavailing and dangerous bleeding persists, as shown by repeated vomiting of fresh blood or profuse bloody diarrhea, resort to surgery.

In cases with continued or repeated slow bleeding sufficient to produce a decided anemia but without immediate danger to life, the failure of medical measures calls for surgical intervention.

Subsequent treatment consists of total abstinence from stomach feeding and drinking for three days, giving saline by rectum every six hours to allay thirst. Give soap suds enema once a day. At the end of three days give milk of magnesia sufficient to clear out all remaining blood from the bowels. Then begin the regular medical treatment for ulcer. Have patient use bed pan for several days after the hemorrhage, and then a commode by the bed.

Chloroma, The Recent Literature and a Case Report. DORSEY BRANNAN (Bulletin of the Johns Hopkins Hospital, March, 1926).

The author reports a case of chloroma occurring in a white male child of fourteen years of age, whose chief complaint was paralysis of the left side of the face, beginning with pain in the ears; followed later by frontal headaches and vertigo. Diplopia soon developed, and vision of the left eye became impaired. His hearing had been poor for some time, and finally his ability to hear was almost lost. There was a swelling on the left temple. Pain over the mastoids developed. Slight exophthalmos was noted. White blood cells 36,800, myeloblasts 86 to 94.5 per cent. He was poorly nourished, and anemic. Bleeding from the gums developed, and the boy became gradually weaker, cachectic and more anemic. The head became unusually large and asymmetrical, resembling somewhat that of a hydrocephalic. Headaches were so severe that he often cried out with pain. The spleen became palpable a month before death. The lymph nodes of the posterior cervical chains, as well as a few axillary and inguinal nodes were enlarged. Pulse rate was usually rapid, and there was an irregular moderate fever. Death took place five months and twenty-six days after admission, ten months after the onset symptoms. The autopsy showed flat, irregular and hard tumor masses, most marked on the left side, scattered over the head. Marked exophthalmos, greater on the left side. Lymph nodes of the posterior cervical triangles, right axilla and in the groin were slightly enlarged, firm and movable. The autopsy findings of extensive green tumors over the skull, in the dura, parabasal sinuses, orbits, along the spine, sacrum and ribs in relation especially with the ligamentous structures and periosteum were distinctive enough of chloroma. Erosion of the bones and pressure atrophy of the tissues by tumor masses and infiltrating cells were well marked. The bone marrow was red and very abundant especially in the femur, but the examination was not extensive enough to exclude possible green patches. The slightly enlarged and firm spleen, the pale yellowish green and enlarged lymph nodes, renal and testicular masses and greenish discoloration of the supporting tissues constituted other but no less important pathological findings. The autopsy findings easily explain the symptomatology. Miscoscopically the tumors, bone marrow, organs and the blood cells showed the same large non-granular mononuclear cells, the myeloblasts, with few or many cosinophilic and a few neutrophilic myelocytes and polymodphonuclear leumyelocytes and polymorphonuclear leucocytes were absent. The infiltration of the organs was essentially the same as that in myelogenous leukemia. The large number of eosinophilic myeloid cells in the lymph nodes and in the edges of the orchitic tumor show the fallacy of associating the green color with these cells. The accumulation of the tumor cells within the lung alveoli was a peculiar feature, but it

has been observed before. The mitoses found were not especially atypical and tumor giant cells were not found. Megalokaryocytes were found only in the marrow. The pathological and clinical findings in this case prove it to be one of chloroma of myelogenous leukemia. This agrees with the better studied cases of chloroma during the last twenty years; they have all been regarded as myelogenous; only, the older cases and recent ones not well studied have been regarded as lymphoid in origin. Many authors have considered, or at least called, all large nongranular, mononuclear cells as lymphocytes, even in the presence of a fair number of myelocytes. Nevertheless, the bone-marrow origin of these cells was recognized more than twenty years ago, and they were called bone-marrow lymphocytes representing predecessor stages of myeloblasts. (This was clearly shown by Warthin in 1904, who was the first to regard *Chloroma as a tumor-like hyperplasia of the parent cells of the leucocytes, primary in the red marrow, the periosteum being involved only secondarily. As a result of this leucoblastic hyperplasia atypical leucocytes* or *leucocytes corresponding to some one of the normal types may appear in the circulating blood in varying numbers.* Chloroma may be leukemic or aleukemic. The present case and all other carefully studied cases since 1904 have borne out the correctness of Warthin's views.) The author concludes that chloroma or chloroleukemia is a myelogenous process, an unusual form of myelogenous leukemia. Aleukemic stages of chlorma are common but true aleukemic forms of the disease are rare. Transitional or border-line and atypical cases occur, which emphasize the close relationship between myelogenous leukemia and chloroma. Nothing new is added to our knowledge of chloroma by this case report; it is only confirmatory of views expressed twenty-four years ago. Nothing new in regard to the green color is given. The writer thinks it probable that this is bound up with the myeloid cells. The peroxidase test was employed in this case in the study of the blood smears and of frozen sections of formalin preserved spleen, dural and cranial tumors, and in each case the reaction was strongly positive. At the present time there is no proof that any case of chloroma is of lymphoid origin, even though the cells in large proportion may superficially resemble lymphocytes. The writer also accepts the neoplastic properties of true chloroma, and regards this point as giving strong evidence of the neoplastic nature of leukemia in general.

Reviews

Physiology and Biochemistry in Modern Medicine. By J. J. R. MACLEOD, M.B., LL.D. (Aberd.), D.Sc. (Tor.), F.R.S., Professor of Physiology in the University of Toronto, Toronto, Canada; formerly Professor of Physiology in the Western Reserve University, Cleveland, Ohio. Assisted by Roy G. Pearce, A. C. Redfield, N. B. Taylor, and J. M. D. Olmsted and By Others. Fifth Edition. 1054 pages, 291 illustrations including 9 colored plates. C. V. Mosby Company, St. Louis, Missouri, 1926. Price in cloth, $11.00.

The fifth edition of this work has been expanded to meet the needs of medical students as a text book of physiology. At the suggestion of various teachers of physiology a section on the physiology of the special senses has been added by Dr. J. M. D. Olmsted, and the section on the neuromuscular system has been expanded by Dr. A. C. Redfield so as to include that part of the subject styled nerve-muscle physiology. The other portions of the volume have been thoroughly revised, and many chapters, such as those on blood, respiration, ductless glands, and the metabolism of the carbohydrates, have been practically re-written to bring the subject matter in line with recent advances relating to these subjects. The book still retains its original purpose of serving as a guide to the application of the truths of physiology in the bedside study of disease. It, therefore, is of value to the clinician, as well as to the medical student. It is, of course, increasingly difficult to present within the confines of a single volume a complete account of the rapidly expanding science of physiology in its correct relationship to previous knowledge, but an attempt has been made through the revision of every chapter to present the reader with at least a fair survey of the science of physiology as it stands today. An examination of the subject matter of the book, to see if it bears out the success of their attempt, shows that it has been very successful in great part, but less satisfactory as far as some subjects are concerned. Particularly well brought up to date are the chapters of the respiration. Gesell's theory that the chemical control of respiration is the hydrogen-ion concentration of the respiratory center is clearly presented, and the author believes that this theory satisfactorily brings into harmony all the previous discordant hypotheses relating to the mechanism. Part VI on the respiration is one of the most up to date and best written portions of the book. Part V, on the circulation, is also very well done. That on digestion is much less satisfactory, particularly the portions relating to liver functions and the bile. A more detailed critical review of the large mass of work recently published upon this subject would be of great aid to practitioner and student. Naturally the chapters on carbohydrate and fat metabolism are thoroughly up to the moment and very satisfactory. Among the less satisfactory portions of the book are the chapters on lymph-formation and edema, renal function, shock, and the endocrinal glands. A wider knowledge of pathology would have helped all of these sections. There is also the usual omission of any section on the physiology of reproduction. Why do physiologists continue with this pre-Victorian silence on this most of all important function?

A Text-book of Pathology. By FRANCIS DELAFIELD, M.D., LL.D., Sometime Professor of the Practice of Medicine, College of Physicians and Surgeons, Columbia University, New York; and T. Mitchell Prudden, M.D., LL.D., Sometime Professor of Pathology, College of Physicians and Surgeons, Columbia University, New

York. Fourteenth Edition. Revised by Francis Carter Wood, M.D., Director of the Pathological Department, St. Luke's Hospital, New York, Director of the Institute of Cancer Research, Columbia University, New York. 1339 pages, 20 full-page plates and 830 illustrations in the text in block and colors. William Wood and Company, New York. 1927. Price in cloth, $11.00.

In the preparation of the manuscript for this, the fourteenth edition, of this well-known textbook on pathology the revising editor has endeavored to add to the text some of the more important developments in the subject of pathology which have appeared in the last few years. Aside from many detailed changes, especially new bibliographic references, the question of vitamins and hormones has been touched upon, changes have been made in the chapters on diseases of the thyroid, pancreas, lungs and blood; and a consideration of some modifications of certain lesions of the liver and gall-bladder have also been included. Additional matter has been inserted in the chapters on infectious diseases, pathology of the bones and joints, and nervous system. A considerable number of new illustrations has been added. The chapter on lesions following various types of poisoning has been eliminated, as these matters are covered much more fully and satisfactorily in the standard works on legal medicine than is possible in a textbook on pathology for students. The chapter on the preservation and staining of pathological tissues has also been omitted. The author would have been wise to have followed his own inclination and have excluded Part III, on the methods of making post-mortem examinations, since it is no longer practical or expedient to include post mortem methods, histologic technique and legal medicine in any textbook on general pathology, as they are in themselves sufficiently large in field and important enough to demand separate treatment, and the field of general pathology is so large that its satisfactory treatment needs the space accorded these other subjects. Also, it is no longer possible to treat in any adequate manner the subjects of General and Special Pathology within the confines of one volume. This new edition is again handicapped from the start in the effort to do this, and the result is unsatisfactory. General pathology demands a broad treatment from the standpoint of general biology, and its satisfactory presentation would require a volume of the size of the present one. Special pathology suffers similarily; in this volume the space allotted to it is wholly insufficient. Although this fourteenth edition is a great improvement over the preceding, it still represents a transition stage between the pathological text-book of the late nineties and the modern conception of a teaching text-book of pathology. It is very difficult to insert new material satisfactorily into old. The patches also show their difference in texture and color, and that defect is very clearly evident in this edition. As it stands it is not a modern text book, thoroughly up to the moment.

Fistula of the Anus and Rectum. By Charles John Drueck, M.D., F.A.C.S., Professor of Rectal Diseases in the Post Graduate Hospital and Medical School, Chicago. 318 pages, 66 original illustrations. F. A. Davis Company, Philadelphia. 1927. Price in cloth, $3.50.

As the author points out in his preface, every general practitioner of medicine is called upon to advise or treat patients suffering from rectal fistulae and many times, if not usually, finds himself confronted by a confusing array of symptoms and conditions with which he feels unable to cope. As a rule little attention is paid in the clinics of our medical schools to this great class of conditions which are by no means infrequent, but on the contrary, constitute a very common class of complaints in the public at large. Because of the numerous and diversified opinions as to the cause and manner of treatment by different authorities. the average physician meets a patient with rectal fistula with some hesitation and trepidation. As a result, improper treatment and imperfect operation are the rule, and most patients, diagnosed as anal or rectal fistula cases, become chronic patients whose end-results are usually

worse than their first condition. Drueck has gathered together the anatomical pathological, physiological and clinical knowledge gathered through years of study of a large number of individuals, who, suffering from fistulae, were required to submit to more than one surgical operation, and even then, many of them were obliged to go uncomfortably through life. In the treatment of fistulae the surgeon must never forget that the technical procedures incident to the removal of the fistula are only the first steps in the treatment, and that he must always preserve the functions of the rectum and anus. In a region, the anatomy of which is so intricate as is that of the perineum, with its many nerve trunks, its multiple muscles, tendons and fasciae acting in different planes, its peculiarly arranged blood supply, the preservation of function presents a very complicated problem, and emphasizes the need of delicate surgical procedures, vigilant postoperative care and subsequent physiotherapeutic measures. The greater part of the volume is directed to diagnosis and to treatment. These chapters are very well written, and the subject well covered and clearly presented. The illustrations are well chosen and are very good in representing the various conditions described. The printing is excellent and there are very few errors. The book can be recommended to the general practitioner as the best and most practical monograph on this subject.

The Evolution of the Tubercle. By Allen K. Krause, M.D., Associate Professor of Medicine and Director Kenneth Dows Laboratories, Johns Hopkins University, Baltimore. 95 pages. Journal of the Outdoor Life, 370 Seventh Avenue, New York. 1927. Price in cloth, $1.00.

The text of this little book was prepared as an address on the research subject "The Anatomical Structure of Tubercle from Histogenesis to Cavity", which was delivered at the Fifth Conference of the International Union against Tuberculosis, Washington, D. C., on October 1, 1926. The author uses "tubercle" in the sense of all anatomical responses to tubercle bacilli, nodular or non-nodular. He traces the development of characteristic forms throughout their evolution and attempts to answer why is *tubercle* the bodily response to tubercle bacilli, how does tubercle behave toward tubercle bacilli, and how does it behave toward the body? He conceives tubercle as of two types—nodular and non-nodular, and gives a morphological description of the origin and early evolution of primary nodular tubercle, and its minute structure. He regards nodular tubercle as representing the native anatomical response of the tissues to tubercle bacilli and non-nodular tubercle as representing a newly acquired allergic anatomical response to tubercle bacilli. In other words his use of the term tubercle is broadly synonymous with tuberculosis, anatomically considered, denoting every kind of formation, nodular, diffuse inflammation, suppurations and effusions of varied kinds, evoked by tubercle bacilli. Using the author's own recapitulation will perhaps give his conception more clearly. Briefly, there are microorganisms, tubercle bacilli, which can live and multiply in the human body, sluggish in development, but hardy of constitution and tenacious of life. Their hardihood and resistance to outside agencies is explained as due to their containing a high content of lipoid substances in the form of a very refractory wax. When living, they settle in the body for the first time, the tissues react to their presence by forming nodular tubercle because they react in this way to the lipoids of the bacilli, the tubercle in effect being a foreign-body reaction serving to protect by walling off tubercle bacilli from normal tissue. Nodular tubercle is a circumscribed process evolving slowly out of the proliferation of cells *in situ*. With the presence of tubercle bacilli in the body, as represented by the establishment of nodular tubercle, the tissues acquire a new and added method of reacting to tubercle bacilli, a reaction to the *proteins* of the tubercle bacilli in the form of tissue *allergy* or *tissue hypersensitiveness*, the *allergic reaction*. This reaction brings about non-nodular diffuse tissue changes characterized by a rapid exudation of cellular elements from the blood to form acute inflammations and effusions. At bottom, tissue

allergy is also a defensive and conservative process. Tuberculous formations may be looked upon as composites of the effects of the lipoids and proteins of the micro-organisms concerned. The first stage, the tubercle, is a foreign body reaction to the lipoids, the allergic reaction a response to the dissociated proteins (tuberculins) derived from tubercle bacilli. Necrosis and fibrosis he regards as sequelae to the allergic acute inflammation of the second phase. The disease tuberculosis is based upon the extension and progression and repetition of tuberculous formations. The essential difference between the disease tuberculosis caused by living bacilli, and a state of tuberculous formations set up by dead bacilli is to be found in the capacity of indefinite and continued development and multiplication of tubercle inherent in living bacilli. In their normal evolution all tuberculous structures come to represent a mixture, in varying proportions, of the effects of nodular tubercle-formation and allergic inflammatory reaction. The ultimate issue of every tuberculous focus turns upon the balance struck between central necrosis and peripheral fibrosis. Necrosis of tubercle is a result of allergy—a sequel of the inflammatory reaction. It begins centrally and extends outward. Fibrosis is brought about through the conversion of epithelial cells of nodular tubercle into fibroblastic types. It is greatly enhanced by the allergic reaction as the latter subsides. It begins at the periphery and extends inward. It is unfortunate that the author has used tubercle as a synonym for tuberculosis, since the essential facts as presented here are concerned with the development of nodular tubercle and its later allergic phenomena. It is evident that this book was written without a broad knowledge of the pathology of tuberculosis. The writer appears to have no knowledge of the essential necrotizing action of tubercle bacilli upon the tissues and cells preceding the development of the foreign-body tubercle. There are many forms of tuberculosis in which the lesion is a local primary coagulation or *caseous necrosis* and in which the foreign-body tubercle never forms and there is no succeeding allergic inflammation or fibrosis. The lecture is written from a too narrow experimental standpoint, and concerns the development of the *local nodular tubercle and its allergic phenomena* alone. It ignores wholly the *primary tissue lesion* produced by the action of living tubercle bacilli within the body, of which there is sufficient pathologic evidence.

Medico-Legal Injuries. By ARCHIBALD McKENDRICK, F. R. C. S., Ed., Medical Referee under the Workmen's Compensation Act; Consulting Radiologist to Edinburgh Royal Infirmary. 341 pages, 65 figures. Edwin Arnold and Co., London, England and Longmans, Green and Co., New York, 1927. Price in cloth, $7.00.

This book was written as the result of a suggestion that an outline regarding accidental injuries, treated in as simple and non-technical manner as possible, to bridge the gulf between the non-medical mind and the highly technical terminology used in the ordinary medical text-books, would be of great assistance to those concerned in dealing with claims for compensation or damages in respect of injuries. It was not an easy task to accomplish this because of the inability to realize to what extent persons outside the medical profession appreciate or fail to appreciate medical terminology, methods of stating problems and drawing conclusions from them. The author has endeavored to make the treatment of each subject as self-contained as possible and this has involved a certain amount of repetition. There are seventeen chapters and three appendices. The chapters treat successively of an introduction to anatomy, the physics of injury, bone and joint injuries in general, regional bone and joint injuries, treatment and after-treatment of fractures and dislocations, back injuries, head injuries, the nervous system and how it works, pain, shock, heart strain, aortic aneurism, rupture of internal organs, accident as a cause of hernia, relationship of disease and injury, rheumatoid arthritis, and bacteriology. The three appendices are devoted to reflexes, guide to nerve supply of muscles of limbs and a glossary of terms. Naturally the chapter on anatomy is exceedingly elementary; that on the physics of injury is good, as are also the next five chap-

ters on various injuries of bones and joints, back and head. The different forms are described in simple language, and the accompanying illustrations serve their purpose. The remaining chapters are too elementary, and there are many important omissions. Fat embolism following trauma receives no consideration, and this is surely one of the most important medico-legal points to be considered in death following bone injuries. Its symptoms are not differentiated from those of shock. Embolism in general is not treated, nor is thrombosis. Neither one is mentioned in the index. A whole chapter is given to rheumatoid arthritis, which the author regards as the third foot of the tripod upon which so many medico-legal claims are founded, the other two being traumatic neurasthenia and shock. American physicians concerned in accident insurance and compensation work will hardly agree to this sweeping statement. One of the most frequently raised questions in the United States in this work is the relationship of trauma to neoplasm, and this important question receives no attention. There is also no consideration of electrical injuries which are so common with us. While this little volume has much of value in it, it is a matter for regret that so much of vital importance has been omitted. It may be recommended for what it does contain.

College News Notes

A good illustration of the activity of Fellows of The College in contributing to medical literature may be taken from the October 22, 1927 issue of The Journal of the American Medical Association. Seven Fellows of The College are contributors to this one issue of The Journal.

Dr. O. H. Perry Pepper, Philadelphia—Hematology of Subacute Streptococcus Viridans Endocarditis.

Dr. John Phillips and Dr. J. P. Anderson, Cleveland—Cardiac Disturbances in Goiter.

Dr. Glenville Giddings, Atlanta—Friedreich's Ataxia in Ten Members of a Family.

Dr. A. F. R. Andresen, Brooklyn—Treatment of Gastric Hemorrhage.

Dr. Kennon Dunham, Cincinnati—The Diagnosis of Tuberculosis in the Child's Chest.

Dr. Julius H. Hess (with Dr. I. McKy Chamberlain), Chicago—Gelatin Added to the Diets of Artificially Fed Infants.

Announcement is made of the appointment by President Smithies of Dr. Clarence Manning Grigsby, 424 Wilson Building, Dallas, Texas, as Governor for the State of Texas to fill the unexpired term of Dr. Kenneth M. Lynch who has presented his resignation due to his permanent removal from that State. Dr. Grigsby is Professor of Theory and Practice of Medicine at Baylor University Medical Department, and is an outstanding man in his State.

OBITUARY

Dr. Peder A. Hoff, St. Paul Minn.

Died during September of heart disease, after a brief illness; aged 57.

Following his graduation from the University of Minnesota Medical School in 1900, Dr. Hoff took a postgraduate course in internal medicine at the Harvard Medical School, and later pursued further post-graduate study at the University of Vienna. He was instructor of medicine at the University of Minnesota Medical School from 1902 to 1914, Visiting physician to the City Free Dispensary 1902 to 1914, a member of the visiting staff of the City and County Hospital 1902 to 1914. At the time of his death, he was staff physician to St. Lukes Hospital, consulting physician to the Northern Pacific Railway and chief medical examiner for the New York Life Insurance Company. During the World War, he held the commission of Lieutenant Commander in the United States Navy.

Several years ago he was elected to membership in the American Congress on Internal Medicine, and subsequently transferred to Associateship in The College. He was also a member of the Minnesota Medical Association, the Ramsey County Association and the American Medical Association.

Dr. Joseph Howell Way, Waynesville, N. C.

Died September 22 of heart disease, at Asheville; aged 61.

Dr. Way was graduated from Vanderbilt University School of Medicine in 1886. Since 1905 he was a member, and since 1911, president of the state board of health, member of the state board of medical examiners 1897 to 1902. During the World War Dr. Way served successively as captain, Major, lieutenant colonel and colonel, being assigned to Base Hospital at Camp Greene, the Rockefeller Institute and the U. S. A. School of Tuberculosis. At the time of his demise, he was colonel, Medical Reserve Corps, U. S. A.

Dr. Way was a member of many medical societies, having acted as president of the Medical Society of the State of North Carolina, past president, secretary and treasurer of the Tri-State Medical Association of the Carolinas and Virginia, member of the House of Delegates to the American Medical Association 1904-09, and in 1909 member of the Reference Committee on Sec-

tions and Section Work. He was elected a Fellow of The College December 12, 1925, and maintained a constant, loyal and helpful interest in its work.

PRELIMINARY PROGRAMME FOR NEW ORLEANS MEETING

Preliminary program of the American College of Physicians for the meeting in New Orleans next spring, beginning March 5. The meeting will start on Monday, at 10 A. M. with the usual address of welcome from the Mayor of New Orleans; the President of Tulane, Dr. Dinwiddie; The President of the Orleans Parish Medical Society and Dr. C. C. Bass, Dean of the School of Medicine of Tulane University of Louisiana. Dr. Frank Smithies will give a reply to the address of welcome. The program will be continued as follows:

1. Dr. Julius Bauer, Vienna, Austria. (Title to be announced).

2. Dr. David P. Barr, "Multiple Myeloma."

3. Dr. L. G. Rowntree and Dr. George E. Brown, "Studies in Blood Volume with the Dye Method."

4. Dr. Joseph Sailer. Title to be announced.

5. Dr. Frank R. Menne, "The Effect of Iodin on the Histopathology of the Thyroid Gland in the Instance of Hyperthyroidism."

Evening Session, Monday, 7:45

1. Dr. Maud Slye, "Cancer and Heredity."

2. Dr. J. L. Goforth, "Natural and Acquired Body Resistance to Neoplasia."

Tuesday Morning, March 6—10 o'Clock

1. Dr. Charles T. Stone, "The Occurrence of Severe Anemia in Myxedema."

2. Dr. W. W. Duke, "Diagnosis and Treatment of the Anemias."

3. Dr. Hilding Berglund, "Liver Diet in Pernicious Anemia."

4. Dr. C. C. Sturgis, Dr. Raphael Isaacs and Dr. Millard Smith: "Treatment of Pernicious Anemia with Liver Fraction."

Symposium on Tuberculosis.

5. Dr. Charles L. Minor. Title to be announced.

6. Dr. F. M. Pottenger, "The Cause of the Varied Clinical Manifestations in Pulmonary Tuberculosis."

7. Dr. Gerald Webb. Title to be announced.

8. Dr. Robert S. Berghoff, "Intestinal Tuberculosis."

9. Dr. John W. Flinn, "A Study of the Differential Blood Count in One Thousand Cases of Active Pulmonary Tuberculosis."

Evening Session—7:45 o'Clock

1. Dr. James S. McLester. Title to be announced.

2. Dr. Allen K. Krause, "The Pathogenesis of Tuberculosis."

3. Dr. T. Z. Cason, "Some Unfinished Research Problems of the South."

Wednesday Morning, March 7—10 o'Clock

1. Dr. L. F. Bishop, "The Pactice of Cardiology."

2. Dr. Morris H. Kahn, "Heart Strain and Its Consequences."

3. Dr. J. P. Anderson, "Discussion of the Diagnosis of Coronary Occlusian with Special Reference to Its Simulation of Acute Abdominal or Other Surgical Conditions, with Illustrative Cases."

Symposium on Epilepsy

4. Dr. H. Rawle Geyelin, "The Relation of Chemical Influences, Including Diet and Endocrine Disturbances, to Epilepsy."

5. Dr. E. Bates Block, "The Relation of Organic Brain Disease to Epilepsy."

6. Dr. T. H. Weisenburg, "The Relation of Extra-cranial Disease to Epilepsy."

7. Dr. Tom Throckmorton. Title to be announced.

Evening Session—7:45 o'Clock

Symposium on Infectious Diseases.

1. Dr. C. E. Birkhaug, "Erysipelas."

2. Dr. A. R. Dochez, "Scarlet Fever."

3. Dr. Jean V. Cooke, Specific Prophylactic Measures in Varicella and Measles."

4. Dr. J. C. Small, "Rheumatic Fever."

Thursday Morning, March 8—10 o'Clock

1. Dr. C. Saul Danzer, "The Pathogenesis and Treatment of Dyspnoea in the Light of Recent Experiments."

2. Dr. T. G. Schnabel, "A High Fat Diet in the Treatment of Migraine."

3. Dr. Henry Wald Bettmann, "Chronic Appendicitis from the View Point of an Internist."

Symposium on Diabetes.

4. Dr. Anthony Bassler, "Chronic Pancreatic Disorders, Diabetic and Non-diabetic."

5. Dr. Frederick M. Allen, "Present Results and Outline of Jeaklic Treatment."

6. Dr. A. A. Herold, "Diabetic Therapy, with Special Reference to the Newer Remedies."

7. Dr. W. H. Olmstead, "The Dietetic Management of the Diabetic in the Doctor's Office."

FRIDAY MORNING, MARCH 9—10 O'CLOCK

Symposium on Tropical Medicine.

1. Colonel Roger Brooke. Title to be announced.

2. Dr. Aldo Castellani. Title to be announced.

3. Dr. Wm. M. Jones, "The Early Lesions of Intestinal Amebiasis."

4. Dr. Bailey K. Ashford, San Juan, Porto Rico. Title to be announced.

5. Dr. Aristides Agramonte, Havana Cuba. Title to be announced.

FRIDAY EVENING, MARCH 9—7:45 O'CLOCK

1. Dr. Julius Bauer, Vienna, Austria. Convocation Address.

The scientific program as given above will take about half of the time of the meeting. The remainder of the time will be occupied by clinics. In the afternoon from 1:45 to 4:30, clinics will he held at the Charity Hospital of New Orleans, Touro Infirmary, the Hutchinson Memorial, the Richardson Memorial, Hotel Dieu, the Baptist Hospital, Mercy and Presbyterian Hospitals. At the Charity and Touro Infirmary, the program will be changed in the middle of the week, so that two separate programs will be put on at these two hospitals. The same will apply to the program at the Hutchinson Memorial. Clinics will be given at the Charity Hospital by Dr. George Bel, Dr. J. B. Guthrie, Dr. Amadee Granger, Dr. A. E. Fossien, Dr. J. L. Lewis, Dr. Randolph Lyons, Dr. J. H. Musser, Dr. Robert Bernhard, Dr. W. A. Love, Dr. George R. Herrmann, Dr. Philip Jones, and others. At the Charity Hospital, Dr. Julius Bauer, of Vienna, will give a clinic on certain days.

At the Touro Infirmary, clinics will be given by Dr. L. R. DeBuys and his staff and by Dr. I. I. Lemann and his staff. Dr. DeBuys' staff will limit themselves to diseases of children; Dr. Lemann and his staff to internal medicine and to subjects related to internal medicine more or less closely, so that it is quite possible with the present plan to have a surgeon to discuss cases in which the internist and the surgeon come into close contact, such as thyroid cases, abdominal conditions and so on.

At the Baptist Hospital, Dr. Oscar W. Bethea will run the program.

At Hotel Dieu, Dr. S. Chaille Jamison, Dr. Maurice Couret, Dr. L. A. Fortier and others will give clinics.

At the United States Marine Hospital, Dr. W. C. Rucker will be in charge of the program. There are always a large number of interesting tropical and unsual diseases at the Marine Hospital.

At the Hutchinson Memorial, clinics and demonstrations will be given daily. Dr. Wm. M. James and Dr. J. J. Vallarino, of Panama, will demonstrate their preparations and x-ray findings in amebiasis. Dr. J. C. Small, in addition to his scientific paper, will give in more detail than can be done at a large scientific meeting the particulars of the studies he has made in rheumatic fever. Dr. Robert S. Berghoff will give an afternoon demonstration on the diagnosis of chest diseases; Dr. T. J. Perkins, "Constitutional Type in Relation to Mental Disease;" Dr. Aldo Castellani, "Fungus Growth;" Dr. C. C. Bass, "Malaria;" Dr. F. M. Johns; Dr. H. W. Butler, "A Slide Method for the Diagnosis of Syphilis;" Dr. Roy H. Turner, "Intestinal Microbiology." A clinical pathological conference will be given one or two days.

The program at the Richardson Memorial will be under the supervision of Dr. J. A. Lanford, who has invited Dr. C. W. Duval,

Dr. Henry Laurens, Dr. Irving Hardesty and Dr. J. T. Halsey, Professors of Pathology, Physiology, Anatomy and Pharmacology, respectively, with their staffs, to demonstrate research problems that they are actively engaged in or which they recently completed.

Reduced Fares

Twelfth Annual Clinical Session, New Orleans, La.

The Executive Secretary advises that all railroads of the United States and of eastern Canada have granted reduced fares to our Clinical Session on the Certificate Plan of fare and half fare. Those who attend the Session will purchase going ticket at local railroad office and at the same time request a "Certificate." This Certificate, when validated by the proper officers at the registration booth at New Orleans, will entitle attendant to purchase return ticket at half rate.

These reduced rates apply not only to the attendant, but to dependent members of his family.

Going tickets may be purchased from March 1 to 7, and the return ticket may be used up to March 13. For those who desire to remain in New Orleans longer, the return ticket may be used until March 24, upon deposit of Certificate with W. H. Howard, Special Agent located in the City Ticket Office of the Louisville and Nashville Railroad, New Orleans, upon payment of fee of $1.00 per Certificate receipt at time of deposit.

The Executive Offices have on hand a number of old YEAR BOOKS, 1923-24. While these have no present value from the standpoint of directory purposes (the new 1927-28 Year Book was issued during the summer), they may be of interest and historical value to some of our members of more recent election. The Executive Secretary will gladly send a copy to any member upon receipt of 50c in stamps to cover cost of handling and mailing.